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No. VII. OF VOL. II., 1935. LONDON, SATURDAY, AUGUST 17, 1935. Pp. 112 - Price 1s.
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ADDRESSES AND ORIGINAL ARTICLES

THE MORBID ANATOMY OF CARIES OF THE THORACIC SPINE IN RELATION TO TREATMENT*

By H. J. Seddon, M.B. Lond., F.R.C.S. Eng.

RESIDENT SURGEON, COUNTRY BRANCH OF ROYAL NATIONAL ORTHOPÆDIC HOSPITAL, STANMORE, MIDDLESEX

A consideration of the treatment of spinal caries must still be based on a clear understanding of the morbid anatomy of the disease. As yet, no specific remedy has been discovered for the treatment of tuberculosis and the proven value of rest and open-air treatment in the cure of joint lesions has not abolished the need for rational mechanical treatment, founded on anatomical and physiological principles.

The morbid anatomy of caries of the thoracic spine is so wide a subject that I propose to refer only to certain aspects of the disease that have received little or no attention in the past. Spastic paraplegia, the most important complication of thoracic caries, has been dealt with recently by Butler and myself [1] and I shall not make more than a passing reference to this condition.

Anatomical Types of Spinal Caries

1. True *tuberculous arthritis* as found in the diarthrodial joints of the extremities, though occasionally seen in the spine, is relatively unimportant. It occurs most commonly in the occipito-atlanto-axial group of joints, and as a secondary condition in the posterior articulations of the cervical, thoracic, and lumbar vertebræ. So far as is known it never occurs as a primary lesion in these small joints.

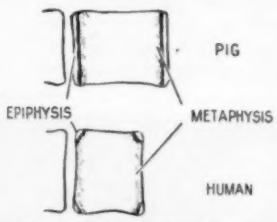

FIG. 1.—Diagram showing vertebral epiphyses of man and pig.

2. *So-called tuberculous epiphysitis* of a vertebral body is a well-known condition, and extremely common in the thoracic and lumbar regions. Strictly speaking, it is not an epiphysitis but rather a metaphysitis. In the typical vertebra of most young mammals the vertebral body is exactly like a long bone. There is a short shaft and two epiphyses. A few years ago the madder feeding experiments of John Hunter were repeated by Prof. J. C. Brash, who has been particularly interested in working out the growth of the mandible.[2] He has allowed me to examine the spines from several of his pigs that were fed on madder from an early age until a few weeks before death. The bones show clearly that all the longitudinal growth in the vertebral body takes place at the metaphyseal regions of the shaft exactly as in a long bone and that the epiphysis contributes little or nothing to the increase in length. In the human vertebra the epiphysis is reduced to an insignificant ring appearing at about the 12th year, and fusing at about the 24th. It contributes nothing to the growth in length, and it never appears to be the primary focus for tuberculosis of the spine (Fig. 1). The lesion is found on the upper or lower surface of the body and is therefore metaphyseal; for this reason the term epiphyseal tuberculosis should be discarded.

An interesting growth phenomenon connected with the metaphyses must also be mentioned. It is sometimes seen in the later stages of spinal caries in children. It has long been known that a prolonged increase of the blood-supply in the neighbourhood of an epiphysis may lead to increased activity of the growth centre. Excessive growth at the lower end of the femur in chronic osteomyelitis of the shaft and in protracted tuberculosis of the knee is familiar to all of us (Fig. 2). A few years ago J. G. Johnstone [3] pointed out that an overgrowth phenomenon may sometimes be observed in spinal caries, though he gave no explanation of the condition. In the cases he reported the rate of bone production in the surviving growth centres of diseased vertebræ was about twice as great as that in the normal vertebræ above and below the lesion. This must be due to long-continued hyperæmia of the growth discs immediately adjoining the lesion (Fig. 3).

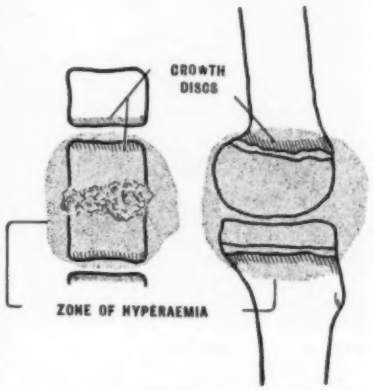

FIG. 2.—Diagram showing how a zone of hyperæmia due to chronic infection may influence adjacent growth-discs.

The earliest X ray sign of metaphyseal tuberculosis is narrowing of the joint space, due to simultaneous involvement of two adjoining metaphyses, and collapse of the intervening disc from interference with its nutrition.

Leriche [4] has stated that the narrowing of the joint space is due to compression of the disc brought about by spasm of the muscles around the lesion. This is hard to believe. The turgidity of the normal disc is so great that it cannot be compressed even by violent flexion of the spine; the vertebral body is crushed before the disc will yield. How could any muscles, however powerful, keep up the terrific force necessary to compress a disc for weeks on end? There is ample anatomical evidence to show that the disc is actually destroyed though not directly by the disease (Fig. 4).

FIG. 3.—Tracings from strictly comparable radiograms of a child's spine showing (on the right) the new bone (shaded) laid down during the course of two years. There has been remarkable activity at the two growth discs adjoining the tuberculous focus.

At this stage, no destruction of bone may be demonstrable radiographically, nevertheless the primary focus is not in the disc but in the bone.

3. A *diffuse osteomyelitis* is also common. In the early stages it is sometimes apparent in the radiogram

* Hunterian lecture delivered before the Royal College of Surgeons of England, 1935.

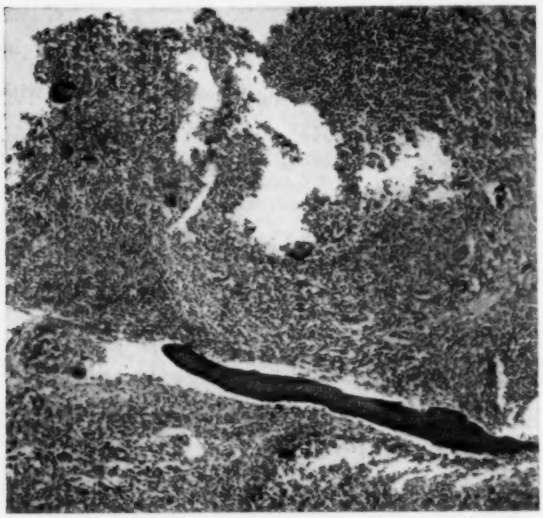

FIG. 4.—Microphotograph of a very early metaphyseal lesion. Tuberculous granulation tissue with giant cells on both sides of an intervertebral disc which is melting away, but shows no signs of actual disease.

as an increase in density of the vertebral body, which is of value in diagnosis. In two cases where secondary foci were suspected in a known tuberculous spine, a diffuse increase in density of two or three vertebral bodies enabled one to localise the new focus. A similar appearance is sometimes found in metaphyseal lesions. It may represent a miliary necrosis of bone with deposition of lime salts. In some cases massive necrosis follows with wholesale destruction of bone; in other cases a more slowly developing rarefaction appears in the usual way. The danger is to mistake this increased density for a sign of healing.

The osteomyelitic focus may lead to widespread destruction of bone within a single body, which may collapse like a concertina, slowly or suddenly. If the collapse is sudden, some of the inflammatory products may be squirted backwards into the vertebral canal and inflict irreparable damage on the cord.[1]

A less malignant infection leads to cavitation of the vertebral body and sometimes the formation of sequestra.

4. Very occasionally the primary focus may appear on the anterior or, less commonly, the posterior surface of a vertebral body—a *so-called periosteal lesion*. It cannot be diagnosed until erosion of the bodies has occurred or until the cord has been compressed—if the disease has commenced under the posterior common ligament.

The spread of a thoracic abscess upwards or downwards causing elevation of the anterior common ligament and erosion of the anterior surfaces of the vertebral bodies adjoining the primary focus is thought by some to be a secondary periosteal lesion (Fig. 5 A). Ghormley and Bradley [5] have pointed out that a collection of pus, tightly compressed between the anterior surfaces of vertebral bodies and the anterior common ligament, may well transmit the pulsations of the aorta to the bone in much the same way as the sac of an aneurysm, and that the erosion so frequently found in tuberculosis is therefore probably of mechanical origin. Four points may be advanced in favour of this suggestion:—

(*a*) The intervertebral discs are often spared, though not to the same degree as in true aneurysm.

(*b*) The aneurysm-like erosion is never found above the arch of the aorta, although anterior abscesses are not infrequent above this level.

(*c*) When an abscess spreads longitudinally under the posterior common ligament such bony involvement as does occur is never of the type found anteriorly—that is, uniform cupped excavation of each vertebral body.

(*d*) In two recent specimens that I have been able to examine there was no tuberculous disease of the eroded vertebræ.

The condition is easily recognised radiographically and it is well to look for it seeing that true involvement of a body may develop anywhere along the course of the abscess. In a number of specimens I have found that the infection has broken in at a higher level—a true secondary focus has formed (Fig. 5 B).

Having defined the four general types of spinal caries, the diarthrodial joint infection, the epiphyseal or rather metaphyseal lesion, infection of the cancellous bone (osteomyelitis), and lastly periostitis, we may now consider the chief forms of the disease as it affects the thoracic spine.

The Typical Lesion

The typical thoracic lesion commences either as an osteomyelitis or a metaphysitis of one or more vertebræ. The tendency to extensive spread of the disease is far greater than in the cervical or lumbar regions, and the reason for this is to be found in the peculiarities of the thoracic spine. In the first place, the thoracic spine is subject to constant respiratory movement much of which takes place between the diseased vertebræ, a point that can easily be verified during the performance of a costo-transversectomy. Many years ago Ménard [6] pointed out that bone destruction in tuberculosis is the result either of *tuberculous ulceration* or *mechanical ulceration*, or both. Tuberculous ulceration is due solely to the activity of

FIG. 5.—On the left is an excellent example of aneurysm-like erosion of several vertebral bodies. (Middlesex Hospital Museum.) I am indebted to Prof. James McIntosh for permission to reproduce this photograph.
In the photograph on the right A is the primary focus. A large fusiform abscess has gradually spread upwards and led to anterior erosion of seven vertebræ and the development of a secondary focus at B. The progress of the condition was followed radiographically.

the disease: mechanical ulceration is the result of movement—friction between inflamed and softened joint surfaces. The thoracic movement in a case of caries leads to mechanical ulceration equalled in severity only by that found in neglected hip disease. The natural thoracic kyphosis also encourages this process. Another factor tending to produce widespread destruction is the close confinement of a thoracic abscess by the strong anterior common ligament and its lateral expansions. In the cervical or lumbar regions, abscesses track away from the spine with the greatest ease. This is rare in the thoracic region, and the extension of a fusiform thoracic abscess may lead to aneurysm-like erosion of many vertebræ and the development of secondary foci above or below the original lesion (Fig. 6 A and B). Where the erosion is purely of the aneurysm type no great damage is done, but the appearance of secondary foci is a serious matter.

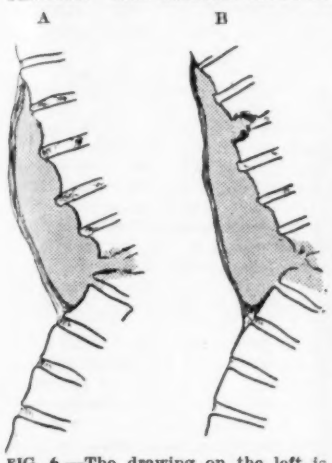

FIG. 6.—The drawing on the left is traced from a radiogram showing an aneurysm-like erosion of four vertebræ above a typical thoracic focus. That on the right shows a similar erosion with a secondary lesion at the upper limit of the abscess cavity. It made its appearance 2½ years after the primary focus was detected.

With further local spread of the disease certain other conditions may arise, concertina collapse and sequestrum formation (to which reference has already been made), and lastly pathological dislocation of the spine. The importance of these three conditions is that they may give rise to severe and often fatal paraplegia.[1]

The evil effects of widespread bone destruction in the thoracic spine are sufficiently grave during the active stage of the disease, but if the patient escapes all these evils his troubles may not be over. Even when the lesion is quiescent or apparently healed further complications may develop.

The Late Complications of Extensive Thoracic Disease

When a number of vertebræ have been destroyed angulation of the spine invariably occurs; the process often begins during the active stage of the disease and may continue for years after the lesion has healed, while the patient is leading a more or less normal life. No form of external support will arrest this bending, and a bone-graft, at least in children, is equally ineffective.

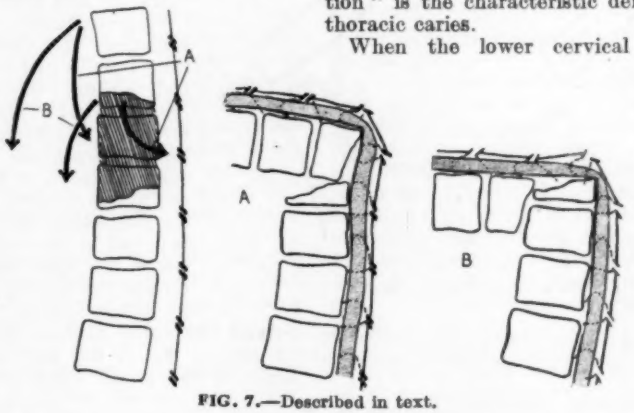

FIG. 7.—Described in text.

If the lesion is not extensive such small gaps as exist during the active stage of the disease are closed ultimately by the apposition of the surfaces of the involved vertebræ. When, however, a number of vertebral bodies have been completely destroyed, the segment of the spine above the lesion swings down through a quadrantal path in such a way that the lower surface of the vertebra above the gap cannot possibly come into contact with the upper surface of the vertebra below. The spinous processes, laminæ, pedicles, and articular processes of the vertebræ that have lost their bodies are responsible for this mechanism. For the sake of simplicity the loss of bone has been represented in the diagrams as confined to three vertebræ (Fig. 7). In actual fact the average number of bodies lost before any of these changes takes place is six. The segment of the spine above the lesion may travel in one of two directions: it may swing posteriorly over the upper surface of the vertebra below the gap (A), or slide down along the anterior face of the lower segment of the spine (B). Let us consider the latter first.

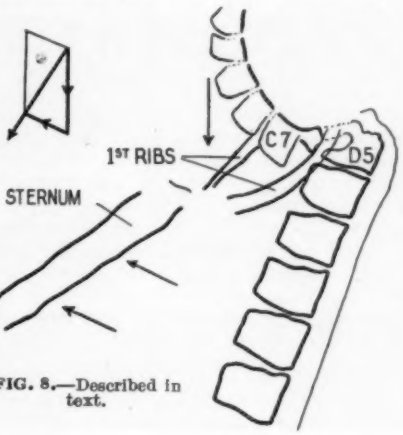

FIG. 8.—Described in text.

ANTERIOR LUXATION OF THE UPPER SEGMENT OF THE SPINE

In all cases where this condition was demonstrated the lesion was situated above the level of the sixth thoracic vertebra. It appears that "anterior luxation" is the characteristic deformity in severe high thoracic caries.

When the lower cervical and upper thoracic vertebræ have been destroyed, the cervical spine has no choice but to luxate anteriorly; it behaves in much the same way as the lumbar spine in spondylolisthesis, the only difference being that in the former the neural arches of the destroyed vertebræ are interposed and compel the cervical column to make its descent with ever-increasing angulation. If the lesion is a little lower—i.e., centred in the upper thoracic spine—an additional factor must be considered. During inspiration in the normal subject the upper end

of the sternum moves very little on account of its firm anchorage to the vertebral column by the short immobile first ribs. If there is a solution of continuity in the upper thoracic spine and the first ribs are attached above the gap, it is possible for the whole sternum to be carried forward during inspiration (Fig. 8); its posterior anchorage is lost and through its attachment to the first ribs it can drag forward the segment of the spine above the lesion. It is clear from the diagram that the interaction of this force and gravity produces the characteristic deformity. Once this state of affairs has arisen, the deformity is likely to progress. The upper and lower segments of the spine can never come into adequate contact, consequently bony fusion cannot occur; indeed, there is often a gap between the two, filled only with fibrous tissue. Spontaneous bony fusion of the posterior elements occurs at the apex of the kyphos, but even if it were supplemented by extensive bone grafting, it is difficult to see how the strongest posterior fusion could support almost the whole of the weight of the head and neck.

POSTERIOR LUXATION

The deformity found in extensive lesions of the lower thoracic region is posterior luxation of the upper segment of the spine.

During inspiration in the normal subject the ribs and sternum are elevated, the antero-posterior and transverse diameters of the thorax are increased. Without describing the mechanism of respiration in detail it must be evident that it is only the relative rigidity of the vertebral column that prevents it from sharing in this otherwise symmetrical expansion of the thorax. If there is a gross defect in the thoracic spine there is no reason why the inspiratory force should not carry back the spine above the lesion in the same way that it carries the sternum forward (Fig. 9). The resultant of this force *In* with gravity *G* follows approximately the same path as that taken by the spine in "posterior luxation," and it is not unreasonable to suppose that this resultant force is the one producing the deformity. Whatever may be the exact cause of "posterior luxation" the frequency with which it occurs after extensive tuberculous lesions in the lower thoracic spine is in no doubt.

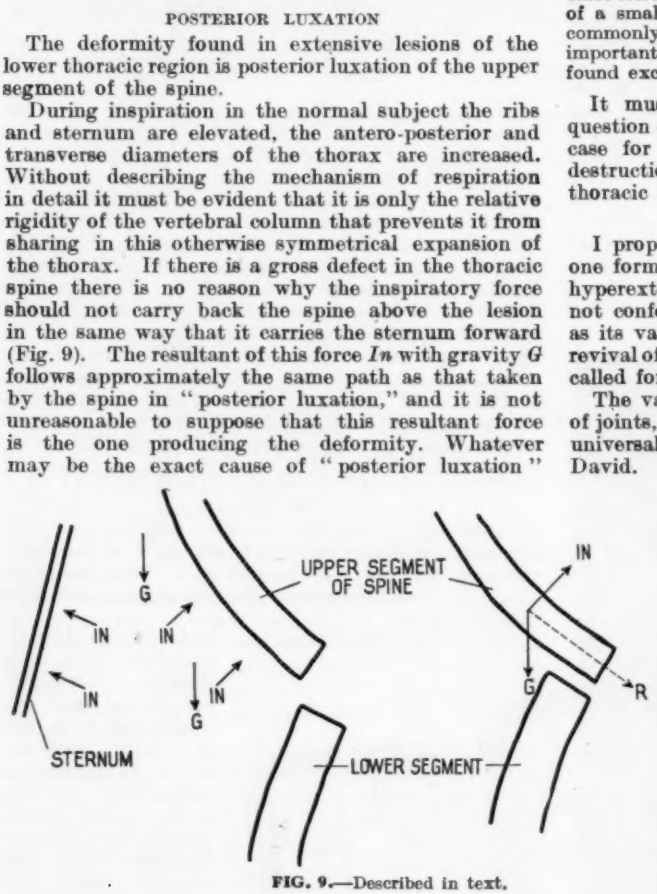

FIG. 9.—Described in text.

The prognosis as regards deformity is better in posterior luxation than in anterior luxation. There is no reason why bony fusion should not take place seeing that good contact between broad bony surfaces is generally present, and solid fusion is often found in spines showing posterior luxation.

Almost everyone has seen patients with severe deformity who lead active and useful lives—suffering, apparently, no inconvenience from marked anatomical changes in the spine and chest. Nevertheless certain serious conditions may arise in cases of gross deformity following thoracic caries.

1. A general lack of development is noticeable in many cases where the disease commenced early in life. In addition to the shortening resulting from the deformity of the spine, there is a definite general dwarfism.

2. There is a diminished resistance to chest infections, and an apparently trivial bronchitis may lead to death at an early age.

3. The cardiac efficiency is sometimes impaired and Carey Coombs has drawn attention to the association of severe kyphosis with sudden fatal cardiac failure.[7]

4. Intractable backache inevitably develops sooner or later in the movable segments of the spine, generally those below the lesion.

5. Lastly, a paraplegia of late onset, developing even 30 or 40 years after the active disease, is not uncommon in patients with severe deformity. In a series of 186 cases of Pott's paraplegia, Butler and I found that half of the cases fell into this group.[1] It is due either to reactivation of a small focus of disease in the spinal canal, or, less commonly, to involvement of the cord by bone. The important point is that late onset paraplegia is rarely found except in cases with gross deformity.

It must now be evident that, apart from the question of the patient's appearance, there is a strong case for every effort being made to reduce bone destruction during the active stage of caries of the thoracic spine.

Treatment

I propose to limit my remarks almost entirely to one form of conservative treatment—namely, rest in hyperextension. It appears to offer certain benefits not conferred by any other type of treatment, and as its value is by no means generally appreciated a revival of interest in this form of treatment is definitely called for.

The value of rest in the treatment of tuberculosis of joints, and particularly the spine, has slowly gained universal recognition since the days of Jean Pierre David. It is only in the methods employed for securing this end that differences are found between one school of surgeons and another. However, rest in hyperextension is a very definite departure from the beaten track, and it is necessary for us to inquire into the origin of this form of treatment.

Hyperextension of the spine in the treatment of Pott's disease first came into prominence towards the end of the last century when Calot introduced his operation of "redressement brusque"—forcible straightening of the deformed spine under anæsthesia. He did not advocate this operation as a form of treatment for the active disease, but rather as a means for correcting the deformity. Calot still believed in the value of rest, though probably not with the fervour that characterised David, Hilton, Hugh Owen Thomas, or Robert Jones. His aim was to hyperextend the spine to such an extent that the line of gravity would fall through the pedicles at the level of the lesion, thus producing stability even though a number of vertebral bodies were absent. We now know that this is impossible, at least in the thoracic region, and his successes must have been due to the production of good compensatory curves above and below the lesion. Calot reported some encouraging results, and such was his enthusiasm

that surgeons all over Europe began to practise his operation.

Then came news of disasters following forcible correction of the deformed spine—sudden death, permanent paraplegia, and recrudescence of the disease being the most serious. Calot finally recognised that his operation, like most others, had its limitations, and he was compelled to modify his original technique to the point of "une douceur extrême." After enjoying a few years of extraordinary popularity "redressement brusque" ended its days in neglect and disrepute, but it led nevertheless to the recognition of a new and valuable principle in the treatment of spinal caries.

Following Calot, Goldthwait, Tunstall Taylor, and many others practised hyperextension in modified forms until finally a new conception of treatment by rest developed: why not nurse the early active case in hyperextension from the first, so that deformity with all its attendant evils could never develop? This was the beginning of the various forms of hyperextension apparatus in use to-day—the frames associated with the names of Bradford, Whitman, and Silver, and the ingenious appliances devised by Gauvain.

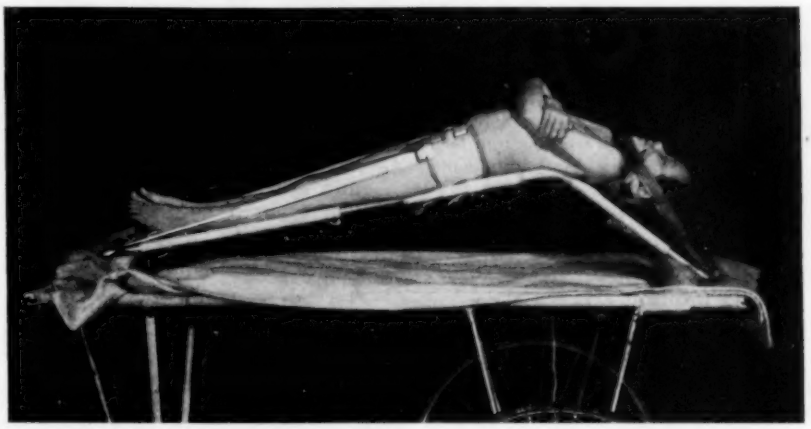

FIG. 10.—The Stanmore frame. The angulation is made exactly opposite the lesion. The pelvic band and head sling prevent longitudinal movement. The frame is no broader than the patient's trunk, and lateral movements are impossible. Almost the whole body surface is available for insolation.

Attractive as this may seem it is, however, a sad fact that a case of thoracic caries diagnosed early and treated from the first by hyperextension may lose several vertebral bodies. A gap in the spine is created, though it may only be demonstrable radiographically. It was the ambition of most of the early advocates of hyperextension to maintain the normal alignment of the spine even though several vertebral bodies might have disappeared during the course of the disease. They hoped to accomplish this during the healing or ambulatory stage by the application of a suitable jacket.

They had fallen into the same fundamental error as Calot, and this mistaken idea is responsible for the strong antagonism to treatment in hyperextension that is found in many quarters to-day. Pott believed that apposition and finally bony fusion of the involved vertebræ was the natural process of healing in spinal caries, and all our experience of tuberculosis of joints goes to confirm his view. Firm ankylosis, preferably bony, is, to use Pott's words, "the thing aimed at." Rest in hyperextension with any other end in view is entirely wrong.

However, rest in hyperextension may be assigned a most valuable place in the treatment of spinal caries on strictly orthodox lines, and I hope to show that it possesses certain merits not found in any other form of treatment.

Let us turn for a moment to the treatment of tuberculosis of the hip. In hip disease, as in spinal caries, bone destruction is the result of the activity of the disease, and of mechanical attrition of inflamed and softened bone brought about by the combined action of muscle spasm, movement, and weight-bearing. Tuberculous ulceration and mechanical ulceration of bone, to borrow Ménard's terms, are present in tuberculous disease of the hip and spine alike.

In the hip, mechanical ulceration can be almost entirely eliminated by combined traction and immobilisation, and Pugh has demonstrated very clearly that strong traction even in the absence of thorough immobilisation reduces bone destruction very considerably.

It is clear that immobilisation combined with powerful traction would be of the greatest possible value in the treatment of caries of the thoracic spine where bone destruction is often so extensive. Fixation does not present great difficulties, but powerful longitudinal traction for any length of time is out of the question. Yet the same effect may be obtained without difficulty, by hyperextension of the spine, provided that constant pressure is applied at the level of the lesion. We must aim at nothing less than the maintenance of a definite gap between the diseased surfaces; only in this way can the constant respiratory grinding be prevented from producing mechanical destruction of the vertebral bodies. That it is possible to produce such a gap is evident from radiograms.

ADVANTAGES OF REST IN HYPEREXTENSION

The peculiar virtues of rest in hyperextension are as follows:—

1. Bone destruction is reduced to a minimum; such destruction as does occur is due solely to the activity of the disease, a factor over which we have only a very limited control. I have been able to compare the bone loss in a number of cases of thoracic caries in children under treatment at the country branch of the Royal National Orthopædic Hospital, one group being treated by hyperextension, the other by simple recumbency. There is no doubt that the average number of vertebræ lost was less in the children treated in hyperextension than in the second series.

It has already been shown that the loss of a number of vertebral bodies results not only in the development of severe deformity of one or other of the types described but that cardiac inefficiency, lowered resistance of the lungs to infection, and late onset paraplegia must all be reckoned among the possible sequelæ of extensive thoracic disease. Treatment in hyperextension is the only measure known which aims

directly at diminishing the incidence of these complications.

2. The accidents resulting from flexion of the spine during the active stage of the disease may all be prevented if hyperextension is begun early. Three conditions have been described: concertina collapse of a single body, the posterior displacement of sequestra, and pathological dislocation of the spine. It is admitted that these accidents are rare, but they are nevertheless of great importance for they are all capable of producing severe and generally permanent paraplegia.

3. The compensatory overgrowth phenomenon in children described by Johnstone has been noted only in cases treated in hyperextension. As this overgrowth has the effect of partially correcting such deformity as is present, everything must be done to encourage its development—another argument in favour of hyperextension. I am not yet in a position to say how frequently compensatory overgrowth occurs, but Johnstone who has devoted a number of years to the treatment of surgical tuberculosis has said that it is by no means uncommon.

APPLICATION OF THE METHOD

What is the best form of hyperextension apparatus? There is no one answer to this question. Any apparatus complying with the conditions already laid down is satisfactory provided that it is used intelligently. I have found that the narrow gas-pipe frame as used by Whitman leaves little to be desired (Fig. 10).

For how long should hyperextension be continued? It is obvious that no change of treatment should be contemplated until there is definite evidence that the disease is becoming quiescent. As in tuberculosis of the hip, our main guide is the appearance of the lesion as seen in radiograms taken at regular intervals of three or four months. One maintains hyperextension until radiography shows, first, cessation of bone destruction over a period of about eight months; secondly, recalcification of the involved vertebræ; and thirdly, improvement in the definition of the bone where erosion has been taking place.

When these signs of quiescence are present (and it may be two or three years from the inception of treatment before they appear) it is time to allow the vertebræ to fall together and fuse. In most cases, in spite of our efforts, several vertebral bodies have been attacked and a definite gap is present. This must be allowed to close; it is the natural process of healing in this disease. The patient should be turned on to his face and nursed in an anterior shell. There is now nothing to prevent collapse at the level of the lesion, and an opportunity is provided for the development of the erector spinæ muscles which suffer considerably when a patient is immobilised for many months on a frame.

When good apposition of the diseased vertebræ has occurred (and it is easily demonstrated radiographically) many surgeons are satisfied that treatment in recumbency has come to an end. A brace is supplied and the patient is discharged from hospital.

But there is another phase of treatment, the development of compensatory curves—a form of treatment that has received more attention on the continent than it has in this country, although the valuable work of Pugh at Carshalton, Waldenstrom [8] in Stockholm, and Calvé [9] at Berck, was recognised long ago.

It is clear that the kyphotic spine tends to become more angulated under the influence of gravity the moment the patient begins to walk (Fig. 11). An increase in angulation means movement at the site of disease and crushing of the involved vertebræ, both extremely undesirable processes. The natural defence against increasing angulation is the development of compensatory lordoses above and below the lesion. Unfortunately the natural curves are often incomplete or even absent, particularly in the thoracic spine, and when such curves do appear they are always long and are segments of large circles. Furthermore, as Calvé has pointed out, and emphasised again recently,[10] the natural compensatory curves not only look ugly but result in considerable shortening of the trunk.

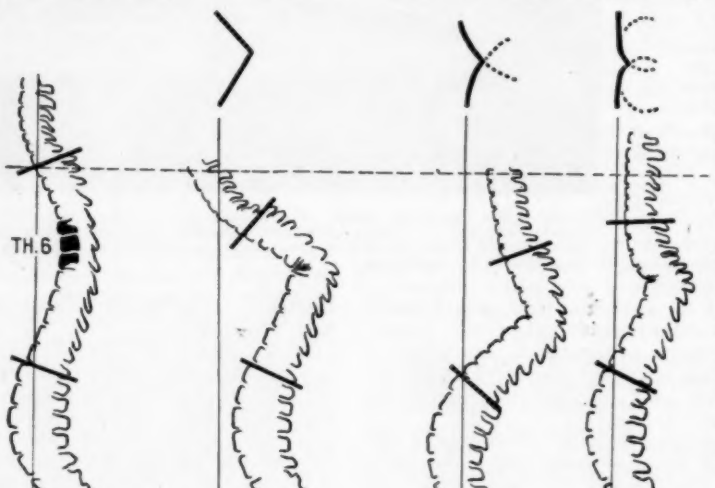

FIG. 11.—(A) Lesion involving body of Th. 6, and adjoining portions of Th. 5 and 7. (B) Uncompensated kyphosis. (C) Natural compensation. (D) Ideal compensation.

The effect of a compensatory curve is to shift the line of action of gravity posteriorly, so reducing the force acting on the arm of the lever formed by the segment of the spine above the lesion.

Butler is strongly of the opinion that an uncompensated spine is always liable to reactivation of the disease and the development of late onset paraplegia, and the case reports that he has collected seem to bear out his contention.[1]

Where bone destruction has been negligible the natural compensatory curves that develop when a patient is nursed on his face after the termination of hyperextension on a frame are quite adequate. In cases where bone destruction has been marked more active measures are necessary: short curves forming segments of small circles must be induced above and below the lesion. This may be accomplished by keeping the patient on the posterior frame, and placing thick pads of suitable material, such as spongy rubber, immediately above and below the kyphos, so as to

produce curves in the most advantageous positions. Where a curve is required in the interscapular region, a central pad is used which presses on the spine between the scapulæ.

The lumbar and cervical curves are only too easy to produce; the difficulty is to produce a marked lordosis in the thoracic region, where one's efforts are resisted by the lack of extension normal to the thoracic spine, and by the rigidity of the thorax. But it can be done.

Let us suppose that the tuberculous focus is quiescent and that adequate curves have been developed above and below the lesion. How is this ideal position of the spine to be maintained? Most surgeons fit the child with a brace, and in many cases it is sufficient. Yet one is tempted to consider spinal fusion as a better form of treatment—a grafting operation designed not only to fix the affected vertebræ and their immediate neighbours, but extending to the adjoining parts of the compensatory curves. Waldenstrom [8] has pursued this policy for a number of years, even in children, and his results are most impressive. If we regard spinal fusion as the most perfect form of spinal support at our disposal, then we have here an admirable field for the operation worthy of wide exploration. Spinal fusion for active disease in children has been tried and found wanting, but it may well be that the operation will prove useful in rendering permanent the good results of conservative treatment.

THE ANEURYSM PHENOMENON

The condition that I have called the aneurysm phenomenon in thoracic caries generally passes unrecognised and consequently the question of special treatment is never considered. Ghormley seems to be the only surgeon who has taken the matter seriously, and he has discussed the possible indication for operation in cases where the condition is present.

He was of the opinion that collapse of all the eroded vertebræ would occur ultimately, even where no true secondary foci had appeared. Costo-transversectomy at an early stage was recommended to empty the abscess, so limiting its longitudinal spread and the depth of the mechanical erosion of the vertebræ adjoining the primary focus. If collapse of the eroded vertebræ does occur, then operation would certainly be justifiable. But in all of the 12 cases that I have followed, there was no suggestion of collapse in any single case. In one case of unusually deep erosion the child has been under constant observation for five years, and there has never been any sign of curvature except at the primary focus of disease. There is, therefore, no justification for interference in the purely aneurysmal type of destruction, and if spinal fusion is contemplated during the later stages of treatment the eroded vertebræ may be regarded as normal for the purposes of operative fixation.

But the possibility of secondary foci making their appearance suggests that the aneurysm phenomenon may after all call for operative intervention, and costo-transversectomy is the only operation that might be of value.

Yet we must remember that secondary foci appear only occasionally, and it would hardly seem right to subject all cases of aneurysmal erosion to costo-transversectomy for the sake of avoiding the secondary foci that might appear in a few cases if the operation were not performed. The conclusion is, therefore, that aneurysmal erosion does not call for any special treatment, but vigilance is required to detect the appearance of secondary foci: routine lateral radiography of the spine is the only means at our disposal for following the progress of this interesting complication of caries of the thoracic spine.

Much of the work embodied in this paper is based on the routine radiographic examination of a large number of cases, and I must express my thanks to my colleague Mr. C. W. Durward for the infinite pains that he has taken in the production of several hundred faultless X ray pictures.

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CONTROL OF WHOOPING-COUGH WITH SERUM AND VACCINE

THE USES OF A NEW SKIN TEST

BY DONALD PATERSON, M.D. Edin., F.R.C.P. Lond.
PHYSICIAN FOR DISEASES OF CHILDREN, GREAT ORMOND-STREET AND WESTMINSTER HOSPITALS

R. H. BAILEY, M.B. Camb.
WANDER SCHOLAR AND REGISTRAR TO THE CHILDREN'S DEPARTMENT, WESTMINSTER HOSPITAL; AND

R. G. WALLER, M.R.C.S. Eng.
ASSISTANT PATHOLOGIST, WESTMINSTER HOSPITAL

THE object of this paper is to give a short review of efforts made during the past ten years to combat and control pertussis, and to put forward (1) a method of preventing the disease in susceptible contacts, and (2) a method of vaccination, controlled by skin test, which affords active immunity.

There are, on an average, 40,000 cases of whooping-cough [1] annually in London alone, and Stocks and Karn [2] maintain that "it is the most important epidemic disease in the great Metropolis in causing physical suffering, loss of school attendance, impairment of physique, and mortality." At its worst it is the cause of between 2000 and 3000 deaths per year in England and Wales.[3] Much work has been done in an attempt to control the disease, as has been done in the case of diphtheria and small-pox, but so far with small success. Methods of treatment are of two kinds:—

(1) Treatment *during an attack*, in an effort to alleviate the symptoms and shorten the course of the disease.

(2) Treatment by *prevention of an attack*, in an effort to immunise the child in infancy.

Treatment with Convalescent Serum

Various remedies have been tried for this acute disease, with little or no success. One worker, however, stands out, in that his line of approach was from a different angle. Jundell [4] in Stockholm aimed at procuring human serum with a high antibody content to treat children already infected with whooping-cough. His method was to raise the antibody titre in the serum of adults, who had had pertussis previously, by injecting three five-daily doses of whooping-cough vaccine, and bleeding them five days later. The serum was then given to the infected child, the total quantity being not less than 60 c.cm. Jundell [4] prefers to treat children in the catarrhal stage, or the first two days of the spasmodic

stage, and in the five cases which he describes in detail all the children had fewer and milder paroxysms than the non-treated control children.

One of us (R. G. W.) working at the Westminster Hospital for the past four years, has made use of convalescent human serum, first in treatment and later in prophylaxis. In the treatment some 25 to 30 consecutive cases, which had been admitted to a fever hospital as clinical whooping-cough, received, on admission, doses of serum varying from 5 to 25 c.cm. No apparent improvement in the clinical condition, such as reduction in the number of whoops per 24 hours, occurred, nor was the total period of illness diminished. It is of interest to note that though these cases were unselected as regards the day of the disease, or severity of illness, none of the patients developed complications, and all recovered.

Prophylaxis with Serum

Since the therapeutic effect of convalescent serum was doubtful, it was decided to try convalescent serum as a prophylactic agent. At the time this portion of the work was undertaken it was not possible to select known susceptible cases on which to practise prophylaxis. Natural immunity was an unknown factor always to be considered.

The ideal method of approach seemed to be the selection of cases in families of children living under conditions of overcrowding. On the first diagnosis of the disease in one child, half the remainder of presumably susceptible children received serum, whilst the other half received no serum. This apparently ideal method did not, however, take into consideration the fact that those who received serum might be naturally immune, or already well advanced in the incubation stage. The results accordingly might well be inconclusive. Further difficulties were encountered, since the number of occasions on which a family was large enough for the satisfactory performance of this experiment was necessarily limited.

Where possible, this method, with controls in the same family, was employed, but there have of necessity been far greater opportunities of attempting prophylaxis in families of two children only. In these cases it was usual to find one child in the whooping stage, whilst the other presented no clinical evidence of the disease. In such cases it was the investigator's practice to give the unaffected child a dose of serum immediately the nature of the disease in the affected child was recognised. Since the incubation period of whooping-cough is a matter of speculation, but is probably in the neighbourhood of 14 days, one is always in doubt as to the stage of incubation which may already have been reached by a presumed susceptible who is to receive serum.

In a series of 95 definite "contacts"—not previously infected, as far as could be ascertained—between the ages of 1 month and 4 years, the majority of whom were not at any time satisfactorily segregated from the source of infection, treated with convalescent serum before there was any clinical evidence of whooping-cough :—

40 (42 %) did not develop the disease.
31 (32 %) developed the disease, but had a "mild" attack.
24 (26 %) developed the disease in an unmodified form.

In a series of "control contacts" in the same age-class, and similarly only partially segregated from the source of infection, and who did not receive serum :—

6 (11 %) did not develop the disease.
11 (20 %) had a "mild" attack.
38 (69 %) developed the disease in an unmodified form.

That is to say, 42 per cent. of patients receiving serum appear to have been fully protected against the disease, but it was not possible to say for certain whether this represents a higher proportion than that of the naturally immune portion of the child population, though it is a higher proportion than in the control series.

In the case of the 32 per cent. who developed the disease in a mild form, it was noted that the catarrhal stage began within ten days of receipt of the serum, and usually within a week. The disease was presumably approximately half-way through the incubation period before serum was administered.

The 26 per cent. of cases developing the disease in the unmodified form were represented by 21 patients to whom serum was given at the end of the incubation period, as evidenced by the fact that the disease was clinically recognisable within two days of receipt of the serum, and by 3 who had received serum early in the incubation period, as evidenced by the fact that the disease was not clinically recognisable until a fortnight or three weeks had elapsed. Again, it is of interest to note that no case developing the disease after serum injection developed complications or proved fatal.

Since this work has been carried out the development of an intracutaneous skin test suggests a modification which may usefully be employed in future investigations, and which would have materially enhanced the value of the preceding work ; that is, the skin-testing of patients before administration of serum with a view to determining whether they are susceptible to pertussis, and after receipt of serum to determine whether they have become immune.

PREPARATION OF SERUM, DOSAGE, AND INJECTION

Blood was collected from convalescents not earlier than one week after all whooping had ceased, and preferably within a month of the termination of the illness. Serum was separated, and the sera from several cases were "pooled." The pooled serum was filtered through Seitz filters, and 0·5 per cent. phenol was added as preservative. The serum was then put up in 10 c.cm. ampoules and kept cool. Various doses have been employed, but it is now the practice to give 10 c.cm., irrespective of age, in one dose, deeply into the gluteal muscles. No untoward "reaction" has been observed to follow such treatment.

Prophylaxis with Vaccines

In 1921, one of us (D. P.)[8] conducted an investigation into 1000 cases, with James Smellie. Actually in 100 cases, those treated with vaccine (first injection containing 500 million, second injection 1000 million, third injection 2000 million, at weekly intervals) took longer to stop coughing and whooping than those cases of whooping-cough which were untreated.

Again in 1931, one of us (D. P.) took part in a large-scale investigation into the treatment of whooping-cough by a fresh vaccine prepared by Bordet himself. The investigation was undertaken by the Ministry of Health, who asked the London County Council, the Great Ormond-street Hospital, and St. Thomas's Hospital to coöperate. The results were uniformly unsatisfactory, those cases treated being as prolonged, or more prolonged, than those cases untreated. Some hundreds of children were injected.

Quite recently Stalling and Nichols [7] have reported good results with an active undenatured antigen.

Sauer [6] attempted to immunise children by the use of a vaccine, and in its preparation he observed that hæmolysis was more pronounced in the cultures of freshly isolated strains than in cultures of long standing. He therefore made a vaccine from fresh strongly hæmolytic strains of the Bordet-Gengou bacillus, grown on human blood for 48 hours, mixed with 0·5 per cent. phenolised physiological solution of sodium chloride. Sauer states that it is diluted

so that 1 c.cm. equals 10,000 million bacilli. He injected 394 selected non-immune children, and gave a total of 7–8 c.cm. (70–80 thousand million bacilli), divided into three weekly injections. During five years the 31 *control children* in 24 of the families contracted whooping-cough, 29 of the injected children were exposed throughout the incubation, catarrhal, and paroxysmal period, but *none* contracted the disease. Although the dose Sauer gives is three times as strong as Madsen [9] gave, no marked local or general reaction occurred, and he believes the immunity is completed in four months, and lasts for years.

The Skin Test

Realising the importance of Sauer's work, and the possible potency of the vaccine prepared according to his directions, an effort has been made to produce a satisfactory test which could be used in the consulting-room to show—

(*a*) Whether a child has had an infection of the Bordet-Gengou bacilli, on some previous occasion.

(*b*) Whether specific treatment in a non-infected child had been of any avail after a course of injections.

To this end various efforts have been made. An agglutination test was first attempted, but proved inadequate, owing to the fact that human serum was used in the preparation of the vaccine, which completely distorted any results obtained, and also the test was considered too elaborate for most medical practitioners to perform. Marshall Findlay (Wellcome Laboratories), working in connexion with the authors, found that on immunising rhesus monkeys with Sauer's vaccine they gave a positive intracutaneous skin test 10 days after the second injection of vaccine. (The first injection of vaccine had been given 10 days previously.)

The vaccine is injected intradermally into the flexor surface of the forearm, the result being read at 24 and 48 hours. A positive test is seen as an area of hyperæmia around the site of the injection, ranging in size from a sixpence to a shilling in diameter. It is transitory in nature, and some experience is required to make accurate readings. It should be seen in 24 and 48 hours, and tends to disappear after this. Just as a marked reaction may be expected in adults with the Mantoux test, the same marked reaction may be expected in adults with this pertussis test, and smaller quantities, even 0·05 c.cm., will give a very definite positive.

THE TEST IN USE

Observations have been made on four groups of children—

(*a*) Seventeen children were seen in the catarrhal stage of whooping-cough, and gave a negative skin-test, but developed a positive skin-test later on in the disease. The following are examples :—

A. B. 3½ years. Cough for 14 days. Skin-tests : May 28th, negative ; June 11th, positive.

C. D. 6 years. Cough began Dec. 11th, 1934. Vomited, whooped. Skin-test done at various intervals was negative. Became positive Jan. 1st, 1935, 4–5 weeks after infection.

(*b*) One hundred children were tested in the ward and at out-patients, some with a history of having had whooping-cough, and others with no such history. Of these, 52 cases gave a positive skin-test, and 48 a negative.

(1) 45 % gave *negative* skin-test and had a *negative* whooping-cough history.

(2) 33 % gave a *positive* skin-test and had a *positive* whooping-cough history.

(3) 3 % gave a *negative* skin-test with a *positive* whooping-cough history.

(4) 19 % gave a *positive* skin-test with a *negative* whooping-cough history.

(*c*) Seven children who gave no history of pertussis, but whose parents desired them to be immunised, gave a negative test before the injections with Sauer's vaccine, and developed a positive skin-test at a later date. The following are examples :—

I

Baby, aged 9 months. Negative skin-test.

Right arm (deltoid).	Jan. 8th..	1·0 c.cm.		
Left ,, ,,	,, 11th..	1·0	,,	
Right ,, (triceps).	,, 15th..	1·5	,,	
,, ,, ,,	,, 18th..	1·5	,,	
,, ,, (biceps).	,, 22nd .	1·5	,,	
,, ,, ,,	,, 25th..	1·5	,,	=8 c.cm.
Intracutaneous test.	Jan. 25th..	Positive.		

No effect on the child was noticed by the mother, except slight fretfulness on the night of the first injection.

II

Boy, aged 2 years 11 months. Negative skin-test.

Right arm (deltoid).	Jan. 11th..	1·0 c.cm.		
Left ,, ,,	,, 15th..	1·0	,,	
Right ,, (biceps).	,, 18th..	1·5	,,	
Left ,, ,,	,, 22nd .	1·5	,,	
Right ,, (deltoid).	,, 25th..	1·5	,,	
,, ,, ,,	,, 29th..	1·5	,,	=8 c.cm.

Skin-test, Jan. 29th, positive ; March 20th, positive.

III

Boy, aged 11 months.

Right arm (deltoid).	Jan. 11th..	1·0 c.cm.		
Left ,, ,,	,, 15th..	1·0	,,	
Right ,, (biceps).	,, 18th..	1·5	,,	
Left ,, ,,	,, 22nd .	1·5	,,	
Right ,, (deltoid).	,, 29th..	1·5	,,	
Left ,, ,,	Feb. 2nd .	1·5	,,	

This child was under observation in Westminster Hospital for the whole course of the injections. After the first injection a rise of temperature was observed, and subsequently on each occasion, but less marked each time.

Skin-test : Feb. 5th, positive.
Blood count : Normal, after course of injections.

Recent inquiry revealed that none of the children who were vaccinated have come into contact with active pertussis up to the present time, nor have they developed the disease.

Technique of Immunisation

The procedure of active immunisation of suitable cases is as follows. It varies slightly from that recommended by Sauer in that three bi-weekly injections are made instead of three weekly ones. Alternate arms are used each time, the deltoid region being injected the first week, and the triceps region the second week, the deltoid region again being used the third week. The dose is—

1st injection	right deltoid ..	1·0 c.cm.	3 days later.		
2nd ,,	left ,, ..	1·5	,,	4	,,
3rd ,,	right triceps ..	1·5	,,	3	,,
4th ,,	left ,, ..	1·5	,,	4	,,
5th ,,	right deltoid ..	1·5	,,	3	,,
6th ,,	left ,, ..	1·5	,,		
Total		8·5	,,	=85 billion bacilli.	

Skin-tests are performed before, during, and after the injection. The skin-tests became positive approximately one week after the last injection. This appears to show that, contrary to what Sauer claims for the vaccine, an allergic state is developed which gives a hypersensitiveness to the skin 3–4 weeks after the initial dose of vaccine, and the child then shows a

positive skin-test. This gives the physician some idea as to whether or not the treatment is effective, and agrees with the result of vaccination in other diseases. Whether or not immunisation is complete then it is difficult to say. Possibly it reaches its height at four months, as Sauer suggests.

Summary

(1) Convalescent serum from cases of whooping-cough has been used with definite benefit in the prophylaxis, but doubtful benefit in the treatment, of pertussis. (2) A preliminary description is given of a skin-test which can be used both to diagnose whooping-cough in an early stage, and also to pick out those children who are susceptible to the disease. (3) The pertussis skin-test is further evidence that children can be immunised against whooping-cough by the giving of a suitable vaccine. (4) Children who give a negative skin-test can be made to give a positive skin-test by the injection of the vaccine.

Our thanks are due to Dr. Marshall Findlay and the Wellcome Laboratories for help with the experimental side of this investigation, and we are indebted to Dr. Findlay for demonstrating the intracutaneous pertussis test experimentally first on monkeys. We are also indebted to Eli Lilly and Co., Ltd., for supplying us with the necessary amount of Sauer's vaccine required in carrying out these tests.

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THE "PREGNANCY" TEST IN RELATION TO DEATH OF THE OVUM

By P. M. F. Bishop, B.M. Oxon.

LECTURER IN CHEMICAL PHYSIOLOGY, GUY'S HOSPITAL MEDICAL SCHOOL; CLINICAL ASSISTANT TO THE OBSTETRIC DEPARTMENT, GUY'S HOSPITAL, LONDON

The practical value of the Aschheim-Zondek and of the Friedman tests for pregnancy is now clearly established. In the diagnosis of uncomplicated pregnancy the tests are extremely reliable and most workers report an accuracy of about 98 per cent. The test has also been employed in the diagnosis of hydatidiform mole and chorion-epithelioma, both of which conditions give very strong pregnancy reactions, as little as 1/2000 of the normal dose of urine being required in some cases to give a positive result. Recently studies have been made on the hormone content of the blood and urine in connexion with certain complications of pregnancy, such as hyperemesis, toxæmia, and eclampsia.

It is therefore natural that the clinician should now appeal to the laboratory worker to assist him in diagnosing death of the fœtus in utero.

Of what value are the biological tests for pregnancy in the diagnosis of intra-uterine death? How long after the death of the fœtus do the tests remain positive? The present communication is an attempt to solve this question. It summarises the results given by the test in a consecutive series of cases in which the date of fœtal death could be determined clinically with some degree of certainty.

The Friedman test was employed in all the cases of the present series. The method of performing the test has been described in detail elsewhere (Bishop, 1933), and will therefore be briefly summarised only:—

An adult doe rabbit which has been segregated for 24 hours is submitted to abdominal laparotomy for inspection of the ovaries. 10 c.cm. of filtered urine for diagnosis is injected intravenously in a single dose. After a minimal interval of 36 hours the ovaries are again inspected by laparotomy. Three possible changes may occur, either singly or in combination: (1) increase in size and prominence of pre-existing ripe Graafian follicles; (2) the appearance of fresh corpora lutea; (3) hæmorrhage into unruptured follicles, the so-called corpora hæmorrhagica. The first of these changes represents a weak positive result. Should either or both of the other reactions occur the result is definitely positive. If none of the changes occurs 10 c.cm. of urine obtained from a woman known to be pregnant are injected intravenously, and again, after a minimal interval of 36 hours, the rabbit is submitted to operation and the ovaries inspected. If the typical changes are present it has been shown that the rabbit will react to urine of pregnancy, and the test can be confidently reported as being negative. If the ovaries still remain unchanged it is obvious that the particular animal employed is resistant to urine of pregnancy, and it is therefore discarded as being an unsuitable test animal, and the procedure is repeated on another rabbit. This modification of the original Friedman technique is designed to eliminate the rabbit as a source of error in the test.

The series is summarised in the accompanying Table. It will be seen that on two occasions (Cases 2 and 5) the test was still positive six weeks after death of the fœtus. Apart from these two cases the test tends to become negative between 10 and 24 days after intra-uterine death has taken place.

Table giving Summary of Cases

No.	Interval between death of ovum and performance of test.	Result of test.	Interval between death of ovum and expulsion or delivery.	Stage of pregnancy at which ovum died.	Condition of placenta and remarks.
1	3 d.	P.	3 d.	7 m.	Placenta 4 lb. Very bulky and pale. Section: active chorionic tissue. Fœtal ascites.
2	6 w.	P.	10 w.	6 m.	—
3	1 w. 3½ w.	P. N.	5 w.	—	— —
4	9 w.	N.	—	4½ m.	—
5	6 w.	P.	—	5 m.	—
6	6 d.	P.	—	—	—
7	14 d. 21 d. 28 d. 35 d.	W.p. W.p. N. N.	52 d.	8 m.	Placenta completely infarcted. Hormone excretion normal at fifth month.
8	10 d.	P.	10 d.	8 m.	Placenta normal. Section: active chorionic tissue.
9	8 w.	N.	8 w.	5½ m.	No gross infarction. Healthy villi. Some degeneration of connective tissue.
10	36 h.	W.p.	6 w.	—	—
11	47 d. 56 d.	N. N.	72 d.	6 m.	Grossly infarcted.

W.p. = weak positive; N. = negative; h., d., w., m. = hours days, weeks, months.

These observations are supported by the reports of other workers (see bibliography). I have been able to find reports of only 31 cases in which the exact

date of fœtal death was determined clinically. In most of these the Aschheim-Zondek rather than the Friedman test was performed, and many of the tests were positive as long as 40 days after death of the ovum. The reason why the Aschheim-Zondek test remains positive rather longer than the Friedman test is because infantile mice are more sensitive to prolan of pregnancy urine than adult rabbits, and will therefore detect smaller concentrations of the material. For instance, in normal pregnancy the Friedman test becomes negative within 48 hours of parturition (Bishop), whereas the Aschheim-Zondek test usually takes about 7 days (Aschheim).

Both Aschheim and Bulliard report cases in which the test was positive three months after fœtal death.

Bué, Palliez, and Gernez describe a case in which the test was positive 10 weeks after the fœtus had died at 3½ months.

Brouha and Hinglais, using infantile male mice as the test animals, obtained a positive result 2 months after death of the ovum.

Stewart investigated a case by the Aschheim-Zondek method, and found a positive reaction 1–2 months after the ovum had died.

Runge and Clausnitzer made very complete quantitative observations on the hormone content of the urine in two cases of intra-uterine death. They report that both the œstrin and the gonadotropic hormone concentrations are considerably less than they are at the same period before normal parturition, but that the œstrin level falls very much more rapidly than the gonadotropic hormone level.

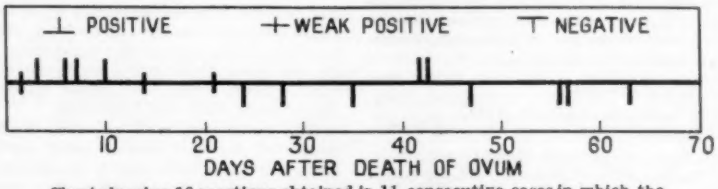

Chart showing 16 reactions obtained in 11 consecutive cases in which the date of the death of the ovum was known.

Westman investigated a case of intra-uterine death of a 7 months' fœtus. Immediately after the death of the fœtus the patient developed rapidly progressive signs of a pregnancy toxæmia. Three weeks after the fœtal movements had ceased to be felt an Aschheim-Zondek test was positive. Six days later a macerated fœtus was delivered by Cæsarean section, and the placenta was examined histologically. Apart from some thrombosis of the stromal vessels the tissue appeared normally active. Implants of the placental tissue were made into infantile mice, as the result of which a fairly strong œstrin reaction developed in the uterine horns, but there was no evidence of ovarian stimulation. From this it appeared that though œstrin was still present in the placenta, the gonadotropic hormones had completely disappeared. Westman suggests that the anterior pituitary had taken over the secretion of the gonadotropic hormones at the time of fœtal death. This would explain the positive Aschheim-Zondek reaction in the urine and the negative reaction with the placental implants. He further postulates that the pituitary may continue to elaborate an excess of gonadotropic hormones for some time after death of the fœtus, so that the urinary Aschheim-Zondek reaction is relatively slow in becoming negative as compared with the rapid disappearance of œstrin.

Spielman, Goldberger, and Frank report a series of 33 cases, but in only 12 of them does the exact date of fœtal death appear to have been clinically determined. In most of the cases both the œstrin and the Aschheim-Zondek tests were performed, the former becoming negative much earlier than the latter, though on one occasion the œstrin test was still positive 23 days after fœtal death.

DISCUSSION

The subject under review is of importance not only from the practical point of view but also from the scientific standpoint. There is sufficient evidence to make it clear that during pregnancy the placenta takes over the function of the anterior pituitary and elaborates a gonadotropic substance, the excess of which is excreted in the urine. That the tissue responsible for the secretion of this factor is the chorionic villus is shown by the fact that in hydatidiform mole the amount of hormone excreted is hundreds of times more than in normal pregnancy. Furthermore, this enormously increased excretion may occur even in the absence of pregnancy, as in the case of chorion-epithelioma, either in the female or in the testis of the male. It would appear, therefore, that the Aschheim-Zondek and Friedman tests depend primarily on the *presence or absence of functional chorionic tissue* and *not on the life or death of the ovum*. Since, however, the main function of the placenta is to provide a means of nourishing the living embryo, one would expect it to become inactive on the death of that embryo. Aschheim states that the factor responsible for a positive "pregnancy" reaction is the biological contact between the maternal blood and the chorion-epithelium; but this is not always true. It is quite common to get a moderately severe post-partum hæmorrhage after the abortion or delivery of a macerated fœtus, showing that there is an adequate maternal circulation in the intervillous spaces, and in some of these cases the pregnancy test at the time of delivery has been negative. For instance in Case 9 the patient had a post-partum hæmorrhage of a pint. Further more, the strength of the pregnancy reaction is not necessarily proportional to the amount of placental tissue. In normal pregnancy the reaction is stronger in the earlier months when the placenta is small and growing than it is towards term, when the placenta is fully grown and bulky. Finally, the activity of the chorionic tissue, as measured by the pregnancy reaction is not necessarily reflected in its microscopical appearance. It is quite possible for the pregnancy reaction to be negative in the presence of apparently healthy villi, as seen under the microscope. This occurred also in Case 9, and on another occasion I obtained a negative Friedman test in a case of retained products, where quite a large piece of placenta had shown perfectly healthy villi in contact with the maternal circulation.

The results cited in this paper make it clear that there is no direct relation between the death of the ovum and the disappearance of gonadotropic substance from the urine, nor should we expect this to be so, on theoretical grounds, for there is no reason to suppose that the fœtus itself produces the gonadotropic factor. Furthermore, the time after the death of the ovum at which the placenta ceases to elaborate the gonadotropic hormone seems to vary considerably in individual cases, and to be determined by no mechanism so far understood. Westman's theory that the anterior pituitary takes over the function of secreting the gonadotropic factor from the placenta immediately after the death of the embryo is interesting, but is not, to my mind, proved by the case which he has studied. In this case 6 days intervened between the collection of the sample of urine which gave a positive Aschheim-Zondek reaction and the implantation of the placental tissue into the infantile mice, which resulted in no ovarian stimulation. Had

a second specimen of urine been taken at the time of the Cæsarean section, and had it again given a positive Aschheim-Zondek reaction, Westman's case might have been more convincing. His theory requires thorough experimental investigation before it can be accepted.

CONCLUSIONS

(1) Where intra-uterine death is suspected clinically, a negative pregnancy reaction will confirm this suspicion; but a positive reaction may still occur as long as three months after the death of the ovum, and cannot therefore be accepted as showing that the fœtus is still alive. (2) A comparison between the œstrin reaction and the Aschheim-Zondek and Friedman tests shows that the former becomes negative considerably sooner after fœtal death than the latter. It may, however, still be positive three weeks after death of the ovum, so that a positive result is by no means certain evidence of a living embryo. (3) The results obtained in the cases investigated throw no fresh light on the factors which regulate the secretion of gonadotropic hormones by the placenta.

I am indebted to Mr. G. F. Gibberd, assistant obstetric surgeon to Guy's Hospital, for the interest he has taken in this work, for supplying me with the clinical details of some of my cases, and for having the placenta histologically examined in a number of the cases.

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THROMBO-ANGIITIS OBLITERANS

(BUERGER'S DISEASE)

GASSING AS AN ÆTIOLOGICAL FACTOR

By J. B. McDOUGALL, M.D. Glasg., F.R.C.P. Edin. F.R.S.E.

MEDICAL DIRECTOR, PRESTON HALL, KENT; AND

J. H. CRAWFORD, M.B. Leeds

ASSISTANT MEDICAL OFFICER, PRESTON HALL

THE following case was referred to us because the patient thought of claiming a pension for thrombo-angiitis obliterans on the ground that it was a disability arising from gassing.

He was a man aged 44 at the time of examination in May, 1934, and he had always enjoyed good health until joining the Army in 1914, at which time he was classed as A1. In 1915 he was severely gassed, and after this he suffered considerably from a troublesome cough and increasing breathlessness. On his discharge from the Army in 1919, he attempted to continue at his former work as a bricklayer but had to give up because of his cough and breathlessness, and in 1922 he obtained a pension for bronchitis as "a permanent disability resulting from his gassing." About this time he developed cramp-like pains in the calf of his right leg, with numbness and coldness of the toes. These symptoms were apparently worse in cold weather and also after resting. With the passage of time the symptoms became progressively more marked, and in 1924 he developed an ulcer under the nail of the second right toe which proved resistant to treatment and became septic, the toe having to be amputated in October, 1924. The amputation wound never healed satisfactorily. The Wassermann reaction of both blood and cerebro-spinal fluid was negative; tubercle bacilli were never found in the sputum and tissue examination for tuberculosis and leprosy also proved negative.

In October, 1928, the third toe of the right foot was amputated for gangrene, and in December of the same year the limb had to be amputated above the knee because of extension of the gangrene up the leg. The amputated limb was sent for full pathological examination and the report confirmed the diagnosis of thrombo-angiitis obliterans. The left leg became involved in a similar manner, and in April, 1931, amputation above the knee was carried out.

On examination in 1934 he was found to be in fair general health only. The pulse-rate taken at the precordium was 84 per minute. No radial pulse could be made out on the right side and the pulse on the left side was only just palpable, and easily obliterated with slight pressure; the vessel wall was somwhat thickened. The blood pressure on the right side was 124/66, and on the left was 140/78. The pupils were equal in size and shape and reacted to light and accommodation. There was no conspicuous sepsis in the teeth or gums. An interesting feature was the presence of multiple lipomata on the right arm and back. There was discoloration and dystrophy of the nails of the right hand with trophic changes at the bases, these being most marked on the little finger where there was also some superadded sepsis. Both hands were cold and cyanosed.

The chest was well covered, with no definite impairment of the percussion note on either side. Breath sounds were a little harsh at the apices, but no adventitious sounds were heard. A good deal of bronchitis was present on both sides of the chest, and even on rest there was a good deal of dyspnœa. The veins of the face and neck were somewhat dilated. The X ray picture of the chest showed no pulmonary infiltration due to tuberculosis, but there was increased translucency over both sides due to a considerable degree of emphysema. The heart sounds were normal except for some accentuation of the second sound at the pulmonary area. Abdominal examination was negative and the urine was normal. The patient has no foreign blood in his veins, and is neither a heavy smoker nor a heavy drinker.

The diagnosis was clearly chronic bronchitis and emphysema following gassing, with thrombo-angiitis obliterans as a complication.

ÆTIOLOGY OF THROMBO-ANGIITIS OBLITERANS

Thrombo-angiitis obliterans was first fully described by Buerger[1] in 1908 and he suggested the name by which it is now known. His monograph on this and allied conditions (the "Circulatory Disturbances of the Extremities," Philadelphia, 1925) is probably the most comprehensive work on the subject. According to him, thrombo-angiitis obliterans is a condition manifesting itself most usually by indefinite or cramp-like pains in the leg, trophic disturbances, and a discoloration of the limb.

As a result of careful study on the pathology of the condition, he described three stages in the progress of the disease.

(*a*) A stage of acute inflammation of the vessels, the inflammatory process involving particularly the deeply situated and larger arteries and veins of the lower and upper extremities, and being rapidly followed by an extensive occlusive thrombosis.

(*b*) A stage of healing or organisation, characterised by the gradual disappearance of the inflammatory products.

(*c*) A final stage in which fibrosis of the adventitia occurred and the vessel was converted into a fibrous cord.

Thus the final result is the obliteration of arteries and veins over a large part of their course by vascularised and canalised connective tissue.

Despite the increasing attention which of late has been paid to this condition, with the subsequent clarification of its pathological features, its ætiology remains obscure. Brown, Allen, and Mahorner [2] show that the cause has been laid successively at the door of tobacco-smoking, the Hebrew race, the male sex, exposure to cold, a shortened coagulation time of the blood, and various nutritional deficiences. Meyer [3] even cites a particular birthplace as a causative factor of importance, believing that patients coming from the belt of land between the Black Sea and the Baltic Sea were particularly liable to this disease.

More important than any of these, however, is the rôle of infection. In Brown, Allen, and Mahorner's series of over 300 cases,[2] the Wassermann reaction was positive in 3 per cent. Evans [4] reports, as leading factors, the occurrence of dental sepsis, nose and throat infections, tuberculosis, prostatitis, typhoid, amœbiasis, and other chronic gastro-intestinal infections. No specific infectious agent has been isolated bacteriologically. Rabinowitz [5] has described an organism which on injection into a rabbit produced an auricular thrombus, but this work has not been confirmed and organisms are not often found in either the acute, subacute, or chronic stages. One is therefore forced to the conclusion that of the many ætiological agents which have been suggested, none fulfils the requirements of a direct causal factor.

A new method of approach to the problem was suggested by Riesman [6] in a lecture on phlebitis and thrombosis. He said, "Barcroft and other English physiologists have shown the fundamental importance of oxygen for the life of the tissues. May not, in Buerger's disease, a lack of oxygen be as important an element in the devitalisation of the tissues as the lack of non-gaseous constituents of the blood?" Meakins and Davies [7] have shown that gas-poisoning provides an example of severe and progressive oxygen-want. These workers have shown that this result, in particular, follows gas-poisoning with phosgene, which liberates hydrochloric acid in the alveoli, with a resultant local necrotic action on the alveolar epithelium and the production of an insidious œdema. Owing to its greater solubility, carbon dioxide passes out more readily than oxygen passes in—and thus a condition of progressive oxygen-want is produced. With a more severe action of phosgene in those exposed to larger and more concentrated doses, there is produced an actual necrosis of the cells with thrombosis of the capillaries. In such cases, oxygen-want becomes well marked. Meakins and Davies [7] have also shown that oxygen-want is a characteristic feature of such conditions as emphysema, chronic bronchitis, pulmonary fibrosis and chronic bronchiolitis—conditions seen in the end-results of gassing.

RELATIONSHIP BETWEEN GASSING AND THROMBO-VASCULAR LESIONS

In emphasising the importance of gassing in the production of thrombotic vascular lesions, the occurrence of such lesions both as an immediate and as a remote result may be noted. Thandavaroyan [8] reports a case of gangrene of the extremity following carbon monoxide poisoning, and a similar case is reported by Briggs.[9] Reidell mentions a case of gangrene of the leg following poisoning with illuminating gas.[10] But on the whole, extensive vascular thromboses as an immediate effect of gas-poisoning are rare, and most of the cases reported are of such lesions as a remote sequel of gassing. Thus Colé,[11] in a study of the remote effects of gassing, is struck by the fact that patients who recovered often showed gangrene of the extremities and various thrombotic tendencies throughout the body. This work was confirmed by Koch,[12] who made a careful study of autopsy material furnished by German soldiers who had been severely gassed and then died some years later. He found that thrombi were particularly liable to form in the femoral veins, the cardiac vessels, and the heart.

The consideration of the formation of thrombi as a particular sequel to gas-poisoning may be taken under three headings.

(*a*) *Alteration in the constituents of the blood.*—The large amount of hæmatin present in the circulating blood after gas-poisoning (particularly after phosgene) is a well-recognised fact, and according to Mayer [13] this is due to the effect of hydrochloric acid produced by the breaking down of the phosgene, as mentioned above. Vedder,[14] in a monograph on the medical aspects of chemical warfare, reports that at the height of gas-poisoning, the erythrocyte count in man was found to be well over 9,000,000. The same polycythæmia occurred experimentally in animals. The peripheral vessels are markedly contracted and the thickened blood flows feebly at the height of poisoning. A similar polycythæmia and also a marked increase in the hæmoglobin percentage was found by Barcroft and his co-workers [15] in their studies of chronic cases of gas-poisoning.

(*b*) *Slowing of the blood stream.*—This is noted as a secondary result to the increase in cellular content of the blood. Buerger [16] considers that the increased viscosity of the blood is one of the most important pathological results of gas-poisoning, and believes that it is partly due to the increased carbon dioxide content of the blood which produces a certain degree of swelling of the red blood corpuscles. Hergt [17] considers that the tendency to thrombosis is due to the increased concentration of the blood (Bluteindickung), the heightened coagulability, and the retardation of the blood flow.

(*c*) *Damage to endothelial tissue.*—The development of pulmonary œdema was referred to in the work of Meakins and Davies, and Gilchrist and Matz [18] have shown this œdema to be due to local injury of the endothelial cells which results in an increased capillary permeability. The actual mechanism in the production of the œdema is due to the increased permeability in association with the acid reaction produced by the hydrolysis of the phosgene.

Thus we see how the three main factors in the production of thrombi—viz., alteration in the constituents of the blood, slowing of the blood stream, and damage to the endothelial cells—are all intimate parts of the sequelæ of gassing.

In the case described, syphilis, tuberculosis, leprosy, gastro-intestinal disease, chronic naso-pharyngeal sepsis, and dental sepsis were all absent. The only factor left which could have produced the pathological changes in the patient's vascular tree described above, was the gassing which he sustained whilst on service, with the subsequent development of chronic respiratory disease, and we are of the opinion that the action of gassing, both directly and remotely, was an effective contributing agent in the production of the vascular lesion.

SUMMARY

1. A case of chronic bronchitis and emphysema following gassing, with subsequent development of thrombo-angiitis obliterans, is described. (2) The general ætiology of thrombo-angiitis obliterans is discussed. (3) The influence of gassing in the production of thrombotic vascular lesions is shown.

We wish to express our gratitude to Dr. L. Buerger, of New York, for his kind coöperation in this case, and for the trouble he has taken, despite his illness, to send us a lengthy personal communication in support of the view which we have advanced.

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THROMBOSIS OF THE PAMPINIFORM PLEXUS

By DONALD McGAVIN, M.B. Camb., F.R.C.S. Eng.

DEMONSTRATOR OF PATHOLOGY, ST. BARTHOLOMEW'S HOSPITAL, LONDON

THROMBOSIS of the pampiniform plexus is a well-recognised result of injury or infection, but its apparently spontaneous occurrence is not so familiar. The condition is certainly not common, for, in searching the records of the surgical unit of St. Bartholomew's Hospital from 1921 to 1934, inclusive, and those of all surgical cases admitted to the same hospital from 1921 to 1928, reports of only two cases could be found. This probably under-estimates its true incidence, since, as it is rarely suspected, a certain number of cases will not have been recorded. The two following histories are reported here not only because well-authenticated examples of thrombosis of the pampiniform plexus are not common, but because the sufferers are liable to be treated by orchidectomy.

CASE 1.—A meat porter, aged 41, was admitted under Prof. G. E. Gask on April 10th, 1928. Five weeks before admission he began to feel pain in his left testicle, and this pain was increased by coughing or by lifting heavy weights, but was not affected by prolonged standing. It became worse during the first few days after the onset, then remained at about the same intensity.

During the five weeks before admission the patient had been kept under observation by his own doctor who noticed a moderate swelling in the left side of the scrotum, which had become smaller by the time he came to hospital. There had been no micturition symptoms, no cough, no night sweats, no weakness and no loss of weight.

On examination there was a hard, nodular swelling, slightly tender, of the globus major of the left epididymis. The body of the testis was normal. The vas was a little thickened but not beaded and there was no thickening of the spermatic cord. On rectal examination the left side of the prostrate was felt to be rather larger than the right, but there was no other abnormality. The patient was apyrexial, his urine was normal and general examination revealed nothing of note.

Although on consultation a diagnosis of tuberculous epididymitis was made, Mr. Paterson Ross, who was the surgeon in charge of the case, said that the short history, the diminution in size, the affection of the head of the epididymis without the tail and the absence of swelling of the left seminal vesicle made him doubtful. Orchidectomy was performed, when the epididymis and the body of the testis were seen to be macroscopically normal. At the lower end of the spermatic cord, in contact with the epididymis, was a mass of fibrous tissue apparently connected with the pampiniform plexus. Microscopical section showed a mass of fibrous tissue surrounding vessels of the spermatic cord. The walls of these vessels were thickened and they contained organised blood-clot. In the epididymis some tubules were normal and some showed in their cells dark brown granules which may have been altered hæmoglobin. There was no evidence of tuberculosis.

The patient was discharged with the wound soundly healed on the eighteenth day after operation.

CASE 2.—A shopkeeper, aged 57, was admitted under Mr. R. M. Vick on Jan. 22nd, 1935. Four weeks before admission there had been a sudden onset of severe pain in the left testicle. The pain radiated from the region of the left anterior superior iliac spine down to the scrotum, but did not go up to the back. There had been no injury. The pain was brought on by standing and was relieved by rest.

The patient had lost one stone in weight during the preceding year. He had no other symptoms of disease. On examination the body of the testis was found to be normal, with normal testicular sensation. The epididymis was moderately enlarged and was hard. The spermatic cord was thickened and nodular. There was no tenderness; no hydrocele was present. Rectal examination showed no abnormality. The patient was apyrexial and nothing of importance was revealed by the general examination.

A provisional diagnosis of tuberculous epididymitis was made, although the patient's age was thought to be against it and the condition was not considered typical. Orchidectomy was performed.

The gross specimen showed the body of the testis to be a little larger than normal. The epididymis and vas were normal, spermatozoa being recovered in large numbers from the latter. The veins of the pampiniform plexus were extensively thrombosed and their walls were thickened and varicose. In microscopical sections the testicle and epididymis were seen to be normal. The veins in the spermatic cord were much dilated and the largest of them was thrombosed, the clot being of varying ages. Beside the oldest part of this thrombosis the vein wall was completely destroyed and in that area there was organisation and recanalisation of the clot. The opposite region of the vein wall showed no abnormality and had no clot attached to it. Most of the arteries showed intimal proliferation and fatty changes. The vas was healthy and spermatogenesis appeared to be normal. The patient was discharged with the wound healed on the ninth day after operation.

DISCUSSION

Although no conclusions can be drawn from two cases, certain points may be noted. Both patients were of middle age, and in both the left side was affected. In neither was there an obvious exciting cause, such as trauma or infection, nor had there been any history of preceding varicocele or an abnormal position of the testicle. In neither case were the seminal vesicles diseased, nor were there firm nodules or softened areas in the prostrate. Active spermatogenesis was proceeding in one case and there was no reason to believe that it had been stopped in the other. Microscopically the walls of the vessels in both spermatic cords gave evidence of degeneration. The pain of one patient was of sudden onset, whereas that of the other began more gradually. The pain of the one was brought on by standing, while that of the other was not affected by standing although increased by coughing or lifting.

The differential diagnoses to be considered depend upon the stage at which the patient is first seen.

If he is examined soon after the onset, epididymo-orchitis is most likely to be simulated, whether gonococcal, coliform or the uncommon acute tuberculous type. Torsion of the spermatic cord may be suggested although in the thrombosis there would not be the associated shock and the testis would be well in the scrotum and not acutely tender. A diffuse hæmatocele of the cord would give a history of sudden onset of severe pain during some effort. Those cases which are seen a few weeks after the onset are liable to be diagnosed as tuberculous epididymo-orchitis. In these the mode of onset, the short history, the absence of any beading of the vas, and the negative rectal examination would be valuable points. The probable freedom of the tail of the epididymis from involvement may also be useful, but a hæmatogenous tuberculous epididymitis starting in the head is a possibility that impairs the value of this criterion. These later cases may occasionally resemble malignant disease of the epididymis. Mr. Russell Howard [1] mentions such a case and he has kindly supplied me with such details as can now be discovered. The man was a patient of Jonathan Hutchinson, junior, and there was an acute onset with symptoms resembling an epididymo-orchitis. After the acute stage had passed a hard lump remained just over the body of the testis. A sarcoma was diagnosed and orchidectomy performed. The specimen is now in the museum of the London Hospital (Genito-Urinary Section, No. 1584), and clinically this case resembled the first history reported in this paper.

The importance of recognising a thrombosis of the pampiniform plexus lies in its bearing upon treatment. Should it be confused with certain other diseases there is a possibility of an unnecessary orchidectomy. This is especially to be avoided, since the microscopical appearances reported here suggest that active function continues in the testicle on the affected side.

I wish to thank Prof. Gask, Mr. Vick, and Mr. Russell Howard for permission to use their cases, and Mr. Reginald Payne for kind assistance.

CONGENITAL MALARIA

WITH REPORT OF A CASE IN ONE OF TWINS

BY N. CECIL TANNER, M.B. Brist., F.R.C.S. Eng.,
ASSISTANT MEDICAL OFFICER, HIGHGATE HOSPITAL; AND
R. F. L. HEWLETT, M.R.C.S. Eng.
ASSISTANT PATHOLOGIST, ARCHWAY GROUP LABORATORY, LONDON COUNTY COUNCIL

ALTHOUGH rare the occurrence of congenital malaria appears to have been proved. Non-congenital malaria in infants is very common; in the hyper-endemic areas of Senegal, M. Adam concluded from his observations that every child was infected by the age of three. Congenital infection has been proved in two ways, either by finding plasmodium in the blood of the child immediately after birth, or by demonstrating it in the blood of the offspring of malarious mothers born in non-malarial districts. In the case to be described the child was born in London, and so comes under the latter heading. A case of congenital malaria occurring in England was recorded in 1924 by Llewellyn Jones and Brown,[1] when a child developed pyrexia, associated with the presence of benign tertian parasites in the blood on the sixteenth day after birth. Our case is of particular interest in that it occurred in one of twins, and we have been unable to find any previous similar record.

[1] A System of Surgery, London, 1932, vol. ii., p. 883.

CASE REPORT

The infant was the second born of dizygotic twins, born at St. Pancras Hospital on Jan. 14th, 1935. The parents were a Bengali, aged 28, and an Englishwoman, aged 25. They went to live in the Rajshi district of Bengal in 1931, and the mother had her first attack of malaria in October of that year. She has had repeated attacks since, the last being in March, 1935. They had five children, the two eldest, aged seven and five, had acquired malaria in Bengal. The third, born in October, 1933, died 12 hours after birth—the mother described it as being "born with malaria, stone cold, but with its head hot and sweating." No blood examination of this child was made. During the whole of this pregnancy, the mother stated that she was never free from malaria.

The mother returned to England in September, 1934, during her last and twin pregnancy. During pregnancy she had one or two attacks of malaria nearly every month, the last being ten days before delivery. She suffered from slight swelling of the feet, but no other symptoms or signs of pregnancy toxæmia. She suffered no abdominal trauma or pain, and there was no evidence of concealed or evident accidental placental hæmorrhage. The labour took place in St. Pancras Hospital and was uneventful; the first born was a male (weighing 6 lb. 7 oz.) and had the fairer complexion. The second, a female, weighed 6 lb. 11 oz., and was darker, with Indian features. Both appeared to be healthy. Obstetric forceps were not used, there was no undue post-partum hæmorrhage, and no bruising of the babies was noticed. No injections of maternal blood were made into the children. Both were breast-fed for 12 days and then bottle-fed.

The female child was admitted to Highgate Hospital under the care of one of us (N. C. T.) at the age of 50 days, with a history of "crying and doubling up its legs" for four days. It had not been feverish, nor had it had any rigors. Though heavier at birth, it was now lighter and shorter than the male twin (it weighed 6 lb. 12 oz.). The child was pale. The spleen was enlarged to 4 cm. below the costal margin.

Blood examination on March 8th, 1935, revealed the following: red cells, 2,310,000 per c.mm.; white cells, 6800 (polymorph, 10 per cent.; lymphocytes, 88 per cent.; large mononuclear, 2 per cent.). Parasites of benign tertian malaria were seen. The van den Bergh test gave negative direct and indirect results. Wassermann and Kahn reactions were both negative. No active treatment was given at this stage beyond the administration of ferri et ammon. cit., grs. 12 per diem. Ten days later blood examination showed: red cells, 4,928,000 per c.mm.; hæmoglobin, 70 per cent.; colour-index, 0·72; white cells, 12,400 (polymorph, 19 per cent.; lymphocytes, 72 per cent.; large mononuclear, 9 per cent.). Malarial parasites (*Plasmodium vivax*) were still present and showed rings, half and three-quarter grown schizonts, and gametocytes. Schüffner's dots were seen in the affected red cells. Three successive examinations of the male twin showed no blood abnormality or parasites. It appeared also to have no splenic enlargement. The mother's blood showed the following: red cells, 5,024,000 per c.mm.; Hb., 90 per cent.; C.I., 0·9; white cells, 5200 (polymorph, 66 per cent.; lymphocytes, 27 per cent.; large mononuclear, 7 per cent.). Parasites of benign tertian malaria were present. Wassermann, Kahn, and gonococcal fixation tests were negative.

Progress and treatment.—During the whole of the time the child has been under our observation there has been a complete absence of pyrexia or rigors. The child was treated with Atebrin, 0·05 gramme daily (in divided doses) for five days, followed by a further course of 0·05 g. daily for four days, after a six-day interval. There was some vomiting and slight diarrhœa during each course, but no other toxic symptoms or skin pigmentation. Films taken on four successive occasions have shown absence of malarial parasites in the blood. On April 23rd the count was as follows: red cells, 5,040,000 per c.mm.; Hb., 80 per cent.; C.I., 0·8; white cells, 8800 (polymorph,

14 per cent.; lymphocytes, 80 per cent.; large mononuclear, 6 per cent.). The spleen also appears to be diminishing in size. The child appears well, and has steadily gained weight (12 lb. on May 10th shortly before discharge from hospital).

DISCUSSION

As this child was born in January, infection by mosquito bite seems to be almost impossible, especially as a mean temperature of 60° F. for at least 16 days must be maintained for *Plasmodium vivax* to develop in anopheles.[2]

In 1910 Beckel[3] recorded 12 cases of undoubted placental transmission of malaria. Griffiths and Mitchell[4] cite several cases described between 1893 and 1928. Thonnard-Neumann[5] reports a case where he found parasites of æstivo-autumnal malaria in the umbilical cord and in the peripheral blood at birth in the child of a syphilitic mother.

Clarke[6] found among 400 placentas that 4·7 per cent. were infected with malaria, but only found parasites in the umbilical cord in one case. Graham Henson[7] made frequent examinations after birth of children whose mothers had been treated for benign and malignant tertian and quartan malaria, but found no parasites in the blood, although they were detectable in the maternal blood just before and after labour. Thibault[8] examined 246 newborn infants whose mothers had active malaria, and found only three with malarial parasites in the blood.

The actual mode of transmission of congenital malaria has not been satisfactorily proved. Blacklock and Gordon[9] found that of pregnant women examined in Sierra Leone, 30 per cent. sustained placental infection, often leading to death of the fœtus in utero or of the child within a week of birth. They never found parasites in the umbilical cord or in the infant's blood, and considered death to be due to absorption of malarial toxins. Wislocki[10] made careful histological studies of the placenta and thought it unlikely that malarial organisms could penetrate the chorionic syncitium. In this instance it is interesting to record that Ascione and Marriotti[11] claimed to produce malarial infection in ten cases by injecting blood (5 cases) and cerebro-spinal fluid (5 cases) from malarial subjects, which had been passed through a Berkefeld V. and N. filter. Panagia[12] suggested that the merozoites might go through a "filterable phase" and then regenerate. If such be true, it is possible that they could pass the chorionic syncitium in such a phase. Zozaya and Sabogal[13] were, however, unable to confirm this mode of malarial inoculation. Such a mode of infection would also be disproved in our case, as only one of twins was affected.

The general opinion is that infection follows gross placental changes or placental trauma at birth. Concetti[14] believes that a placental infarct is the antecedent. Forbes[15] thought that some abnormality of gestation or delivery could be presumed as the parasite passes the placenta so rarely. It appears that malaria in the mother need not be active in order to give birth to an infected child (Beckel and Forbes).

CONCLUSIONS

In the case which we are reporting, where the first child of twins is non-infected and the second has malaria, it might be postulated that after the birth of the first child and the commencing separation of its placenta, the uterine contractions would have caused some separation of, and damage to, the fastening villi of the second placenta before the birth of the second child, and thus enabled parasites from the maternal blood sinuses to enter the child's circulation. We think that the absence of typical bouts of temperature and rigors may be explained by a transferred immunity from the mother during pregnancy.

SUMMARY

(1) A case of congenital malaria in one of twins is recorded. (2) Treatment with iron, without specific drugs, rapidly improved the anæmia. (3) Treatment with atebrin seems to have cured the malaria. (4) It appears that, in the absence of a known placental trauma or infarct, premature placental separation was the most probable cause of the infection.

We wish to acknowledge our indebtedness to Sir Frederick Menzies, county medical officer, for permission to publish this case, and to Dr. P. Manson-Bahr for his help and advice.

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THE EFFECT OF SUDDEN SEVERE ANOXÆMIA ON THE FUNCTION OF THE HUMAN KIDNEY

By R. A. McCance, M.D., Ph.D. Camb., F.R.C.P. Lond.

ASSISTANT PHYSICIAN IN CHARGE OF BIOCHEMICAL RESEARCH KING'S COLLEGE HOSPITAL, LONDON

In human nephritis the excretion of urea, creatinine, and ammonia are usually simultaneously impaired. Following diabetic coma, however, as McCance and Lawrence[1] have shown, the kidney may temporarily become disorganised, so that the excretion of urea and creatinine may be practically in abeyance, while that of ammonia may remain quite normal. The present paper records another example of selective functional disorganisation.

The subject of these observations was a healthy male aged 36. For experimental purposes he had on four previous occasions overbreathed for 45 minutes, and did so for the fifth time on the day in question. Everything went according to plan until the subject had finished overbreathing. Then, either from carelessness or over-confidence, he did not voluntarily begin to breathe again as he had intended to do, and as he had always done before. Acute anoxæmia inevitably followed and was sufficiently severe to cause unconsciousness for about eight minutes. For the next two hours the subject was in a very collapsed state. He sweated profusely for a few minutes and began to feel very sick. He lay in an easy chair for about an hour and then, on attempting to get up, vomited much of the water which he had drunk

TABLE SHOWING RESULTS OF ANALYSIS OF FIVE SPECIMENS OF URINE

Time and events.	(1) 2.45–3.45. Before overbreathing.		(2) 3.45–5.10. Overbreathing, 45 mins., then anoxæmia and unconsciousness.		(3) 5.10–6.20. Period of collapse. Vomit at 6.15.		(4) 6.20–7.30. Still collapsed. Return home by taxi. Hot bath.		(5) 7.30–10.14 bed. Recovery. Substantial meal at 9.30.	
Appearance of the urine	Normal.		Normal.		Dark, turbid and pigmented.		Dark and pigmented.		Normal.	
Urine c.cm. per minute	1·22		1·32		0·25		0·25		1·45	
Urea clearance ($U\% \sqrt{v}/B\%$) as per cent. of standard normal	107		49		17		33		114	
	A*	B*	A	B	A	B	A	B	A	B
Urea	1·28	15·6	0·56	7·4	0·47	1·20	0·88	2·16	1·25	18·0
H_2SO_4	0·126	1·54	0·110	1·4	0·376	0·94	0·404	1·00	0·101	1·46
Creatinine	0·115	1·40	0·075	1·00	0·42	1·04	0·435	1·06	0·089	1·29
Na	0·620	7·5	0·413	5·45	0·340	0·84	0·395	0·97	0·266	3·85
K	0·475	5·8	0·475	6·2	0·515	1·29	0·600	1·48	0·198	2·90
Cl	1·28	15·6	0·375	4·9	0·480	1·20	0·760	1·88	0·320	4·65
P	0·062	0·75	0·033	0·43	0·050	0·13	0·034	0·085	0·049	0·71
pH	6·8		7·4		4·8		4·9		6·8	
NH_3 (as c.cm. N/10 acid/100 c.cm. urine)	12·2		8·5		86·0		86·0		12·7	
Titratable acidity (do.)	11·0		5·3		55·0		54·0		11·0	
$NH_3 \times 100/NH_3$ + titratable acidity	53·0		62·0		62·0		62·0		53·0	
NH_3 coefficient ($NH_3N \times 100$/urea N + NH_3N + creatinine N)	2·6		4·0		24·0		17·0		2·8	

*A = grammes per 100 c.cm.; B = rate of excretion mg. per minute.

during the course of the experiment. Feeling a little better after this, he moved about the laboratory for a short time, went home by taxi, had a hot bath and got into bed. During all this time he felt intensely cold and miserable. Soon after getting into bed he began to feel better and after about 1½ hours to feel hungry. Accordingly he ate a substantial meal and went to sleep soon afterwards. On the following morning he was normal in every way.

Five specimens of urine were obtained and analysed, and the times at which they were obtained and their relation to the salient events are recorded in the Table. It is unfortunate that no record of the blood pressure was obtained, but the subject, who was himself in charge of the experiments and the only person present likely to realise its significance, omitted to have this measurement made. The oversight however does not in any way invalidate the reported observations. In the Table also will be found a record of the analytical results.

The methods used have been those described by McCance and Lawrence [1] except that (*a*) the urea in both blood and urine was determined by modifications of Beattie's method [2] which is a colorimetric xanthydrol procedure. (*b*) The ammonia was separated from the urine in Conway and Byrne's apparatus [3] and determined colorimetrically by Nessler's reagent. (*c*) The sulphates were determined by Fiske's [6] method.

It will be noticed that the volume of urine became very small during the period of collapse and that the percentage of urea in the urine fell to about one-third of its original figure. In consequence the urea clearance (van Slyke's formula) fell from 107 per cent. of normal, before overbreathing, to 17 per cent. of normal during the period of collapse. A fall in the urea clearance of this subject during overbreathing had been observed before, but on previous occasions the clearance rose again at once. That it did not do so on this occasion but fell still further seems to have been the direct result of the anoxæmia. The clearance remained subnormal for nearly 3 hours and then rose to 114 per cent. of normal. Since no determination except that of urea was made upon the blood, no other clearances can be discussed. It will be seen from the Table, however, how "rates" of excretion were affected. The rate of excretion of urea fell to about one-fifteenth of its original figure, while that of sulphate fell only to about two-thirds. The excretion of creatinine was still less affected. The rates of excretion of sodium, potassium, chlorine, and phosphorus fell to values between one-tenth and one-fifteenth of their initial figures. The relative rates of excretion of the ions was no doubt altered to some extent by the rapid changes of pH in the urine, but the general reduction is obvious. The fall in phosphorus may have no bearing on renal function after anoxæmia, since a fall has been observed to follow overbreathing on other occasions. Turning to the excretion of ammonia, all the evidence goes to show that this was not impaired by the anoxæmia. During the period of collapse the pH of the urine fell to 4·8 and the titratable acidity rose to ten times its previous value. The percentage of ammonia in the urine increased even more and the ratio NH_3/NH_3 plus titratable acidity actually rose a little after the anoxæmia. At this time also the "ammonia coefficient" rose to the remarkably high figure of 24. If, in fact, one assumes this ammonia to have been made from urea, the very great fall in the rate of urea excretion (and the urea clearance) is in part explained. Further, the changes of urinary pH show that the kidney had lost none of its capacity to concentrate hydrogen ions. This is true also of the kidney after diabetic coma.

The theoretical interpretation of all these observations is at present rather obscure because not enough is known of the way in which urea, creatinine, and sulphates are normally excreted, and particularly of how the clearances of these three substances are related to the true glomerular clearance. The observed falls in the excretion rates of creatinine, urea, sulphates, and possibly the other salt ions, coupled with the probability that some fall in blood pressure took place, indicates some fall in glomerular filtration. Since however one cannot assume that the change in the rate of excretion of either creatinine, sulphates, or urea is proportional to the change in the rate of glomerular filtration, especially under circumstances such as these, closer analysis becomes

extremely difficult and would be of little value at this stage.

On the other hand, the excretion of ammonia is known to be a special function. The process is stated to be highly susceptible to poisoning by uranium.[4 5] The present results show that, apart from diabetic coma,[1] ammonia excretion may be left unaffected by agents which interfere with the excretion of many of the other urinary constituents.

SUMMARY

After overbreathing and accidental anoxæmia with unconsciousness it was found that: (1) The process of ammonia excretion and the ability to concentrate hydrogen ions were totally unaffected. (2) The rates of excretion of creatinine and of sulphates were reduced respectively to three-quarters and two-thirds of their initial values. (3) The rate of excretion of urea was reduced to one-fifteenth of its original value. Part of this reduction may have been due to the great increase in the excretion of ammonia. (4) The rates of excretion of water, sodium, potassium, chlorine, and phosphorus were reduced to between one-fifth and one-fifteenth of their original values. (5) Recovery was apparently complete in three hours.

The author is indebted to E. M. Widdowson, L. R. B. Shackleton, and R. J. Millar for assistance with some of the determinations.

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Clinical and Laboratory Notes

ERYTHROMELALGIA AND PROCTALGIA FUGAX

By H. S. Carter, M.D. Leeds, D.P.H.

SENIOR ASSISTANT BACTERIOLOGIST, PUBLIC HEALTH DEPARTMENT, GLASGOW

THE paper on "proctalgia fugax" by Dr. Hess Thaysen in THE LANCET of August 3rd, 1935, reminds me of a patient I attended when I was in practice in 1920, whose symptoms were unusual and interesting. On notes of the case then taken I draw for this account.

Mrs. A., aged 52, complained of paroxysmal pain in the right foot with redness and swelling of the dorsum and toes. She had had five or six such attacks during a period of three years, the attacks lasting from three weeks to two months, during which times she had severe pain on many occasions lasting from a few minutes to about an hour. Between the periods of illness the foot gave no trouble; during the bouts, even when the pain was not active, the foot did not feel quite normal and, though not obviously swollen, felt "sort of numb and too big for my shoe." This condition of affairs, I was told, had variously been diagnosed as gout, erysipelas, and inflammation of the foot. Being at that time in the throes of the composition of a thesis on causalgia, I had a mind above common diseases, and boldly diagnosed erythromelalgia, which in the light of a little more wisdom I think was correct.

The earlier attacks had consisted solely of pain in the toes and anterior part of the sole of the foot, and the redness and swelling had supervened in later attacks. Heat aggravated the condition, and she had been worse in South Africa whence she had come, than since she had been in our cooler climate. She could not bear to be near a fire during the attacks, most of which, though not all, had occurred in hot weather. The pain was an intense burning pain accompanied by sensations of throbbing, and worse when the leg was dependent. She got most comfort by lying down, elevating the leg on a pillow, and covering the foot with cloths soaked in cold water. The redness during a paroxysm, as I saw it, was quite bright and affected chiefly the great toe and little toe and the outer side of the foot. There was nothing above the ankle. As an attack subsided and the pain diminished, the skin of the foot was hyperæsthetic, stroking or gently scratching giving rise to unpleasant paræsthesiæ, and the colour of the skin became a curious pale mauve or heliotrope. No hyperidrosis, thinning of the skin, delayed desquamation, deformity of the nails or other trophic change was observed. During an attack the foot felt hot, the vessels were distended, and there was distinct swelling without pitting.

It was in the course of the treatment of this disability during an exacerbation lasting about six weeks that the disconcerting symptom of rectal pain appeared. She had four attacks of this, three in the daytime and one at night at intervals of several days. The duration was short, and on the two occasions I was sent for the pain had subsided by the time I arrived. Her description of it was graphic enough. She experienced a slowly increasing sense of fullness about four inches up in the rectum, as though there was "a small ball expanding slowly." This sensation of expansion or tension soon turned to pain which increased to an intense ache in a few minutes and which at its zenith became almost unbearable, and caused profuse local sweating of the nates sufficient to leave a damp area on the clothing nearest the skin. This pain never lasted more than five minutes at most and subsided fairly quickly, leaving a feeling of weakness and comparative exhaustion. No associated sensations in the præcordium or epigastrium as described by Dr. Thaysen were complained of, though the exhaustion after the attack amounted to a feeling of faintness. There was no accompanying feeling of tenesmus or passage of flatus. The whole attack was over well inside fifteen minutes. This rectal pain never occurred simultaneously with a paroxysm in the foot.

The patient was a woman of average build in good general health who had just passed through an easy climacteric. She had had one child with easy labour and no pelvic complications or sequelæ. There was no history of intestinal illness such as dysentery. A rectal examination revealed nothing. There were no hæmorrhoids and no trouble with defæcation or micturition. The urine was normal, the blood pressure 148/95 mm. Examination of the heart, lungs, and abdomen yielded no help, and there were no signs in the nervous system suggesting syringomyelia or disseminated sclerosis or tabes. The fundi appeared normal. Unfortunately the Wassermann tests on the blood and cerebro-spinal fluid were not carried out, but there was no history or evidence of earlier syphilis. In short, nothing was found to account for the distressing symptoms, which I did not understand.

The attacks of rectal pain were four only and did not recur at that time, and the erythromelalgia becoming quiescent I ceased to see the patient and have no further history. The whole condition was treated with belladonna, valerian, and bromide, with aspirin during the attacks of pain in the foot, together with applications of menthol and cold to the foot. No special treatment was directed at the rectal pain for which no specific cause was found and of which the evanescence probably impressed me more than its severity which I never witnessed. I have thougbt since that it might have been one of those painful spasms of tubular structures which are far from uncommon in neuropathic individuals.

The coexistence of the conditions erythromelalgia and proctalgia is interesting and rare and suggests a common origin. I have since seen a second case of erythromelalgia in a war pensioner, but he had no rectal pain. The ætiology of "red neuralgia" is obscure but it has been suggested that it may originate in an affection of the vasomotor centres in the cord.

Possibly some minute disturbance in the region of the lower lumbosacral roots may account for these phenomena. The equally intense and similar pain of causalgia (thermalgia) may be caused by a microscopic non-progressive lesion of a peripheral nerve. A similar lesion in another place may account for erythromelalgia and proctalgia, contemplation of which provides an interesting speculative study in neurological localisation.

A CASE OF PSITTACOSIS

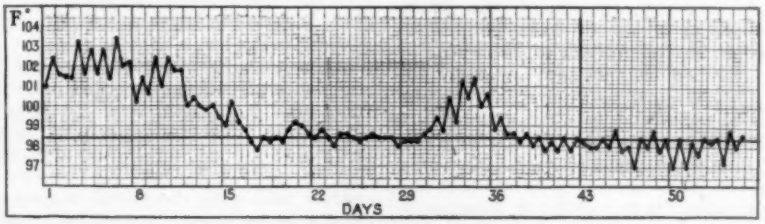
By Smeeton Johnson, M.D. Lond.

Hon. physician to the general hospital, kidderminster

With a note by

Walter Levinthal, M.D. Munich

Psittacosis or parrot disease became widespread in Western Europe in 1929–30, and the importation of parrots and parakeets has been prohibited in certain countries. It has been suggested that the budgerigar, which is less susceptible to psittacosis than the parrot, may become a carrier of the disease,[1] and this suggestion lends interest to the following case.



Temperature chart from May 15th.

The patient is a man aged 63. He has a study in his garden connected to his house by a covered glass passage, which for the last eighteen months has been used as an aviary for 10–14 budgerigars. Consequently he has walked through the aviary several times daily. No particular illness has been noticed amongst the birds, but two or three have mysteriously died. These were purchased from two independent dealers earlier in the year.

On May 15th, 1935, the patient was seen for the first time. He gave a history of headache, pains in the back and limbs, and general malaise of twenty-four hours' duration. He was emotional and evidently felt very ill. His temperature was 101° F., pulse-rate 90, respirations 20, and his tongue was moist and coated at the back. There were no other signs of disease. On the third day of the illness an area of dullness was discovered below the angle of the right scapula. Breath sounds were diminished and heard with difficulty in this region, but there was no cough or expectoration and no pain. The temperature was maintained at 101–3°, with pulse-rate 90 and respirations 24. There was profuse sweating at night and constipation. The patient had intense headache of a throbbing character, stuffiness of the nose, and occasional slight epistaxis. He was apathetic and took little notice of his surroundings, but roused up when spoken to. At the end of the first week the symptoms were much about the same, but he was more apathetic and drowsy. The patch of dullness in the right chest was still present, and there was almost complete absence of breath sounds in this region. There was no abdominal distension or enlargement of the spleen, and there was no rash.

The Widal reaction on the ninth day of the illness was negative to typhoid, paratyphoid A and B, and *Bacillus melitensis*. Bacteriological examination of the fæces was also negative. On May 24th a specimen of sputum was sent to Dr. Walter Levinthal, at the National Institute for Medical Research, Hampstead. As a result of his tests, which are described later, he was able to demonstrate the presence of the psittacosis virus.

On May 30th crepitant râles were heard in the right chest in the scapular region, and cough and expectoration became more frequent, but were never profuse. The temperature reached the normal line and the patient felt very much better. Convalescence continued, and on June 11th, all symptoms having disappeared, the patient was allowed to get up.

A specimen of blood was sent to Prof. S. P. Bedson at the London Hospital on June 13th. Prof. Bedson later reported that the serum gave a positive reaction with the complement-fixation test for psittacosis.

On June 14th the patient complained of pain in the right scapular region. Examination revealed an area of dullness corresponding with the original patch of dullness, and there was absence of breath sounds. There was occasional cough which was painful, and expectoration of mucus streaked with a little dark blood. The temperature was 100°, with pulse-rate 90 and respirations 24. The temperature continued to rise for three days and then gradually fell to normal on the seventh day. On the fourth day the pain had practically gone, and on the sixth day breath sounds began to return, accompanied by fine crepitant râles. There was no severe headache, and perspiration was not excessive.

This case at first resembled a typhoid septicæmia. Certain features, however, distinguished it from the typhoid group of diseases. The onset was sudden and the temperature rapidly rose to its maximum. Headache was intense and perspiration profuse, necessitating the changing of the night attire two or three times a night. Constipation was the rule throughout the illness, and there were no other abdominal symptoms. The spleen was never palpable and there was no rash. During the height of the fever the patient was apathetic and drowsy, and on one or two occasions only was there a little muttering delirium.

The lung symptoms were certainly unusual. The toneless dullness, with very faint or absent breath sounds and normal or slightly increased vocal resonance, and the absence of pain, cough, and expectoration, gave a picture very different from that of lobar pneumonia. Evidently there was some consolidation of the lung, probably associated with obstruction of the smaller bronchi. Resolution was accompanied by fine râles. The fever was maintained for two weeks and subsided to normal during the third week. Throughout the illness the pulse-rate was between 80 and 90, and the respirations were never more than 24. The rise in temperature on June 14th and the return of the lung symptoms were evidence of a mild relapse.

Experimental demonstration of the virus of psittacosis in the sputum by Dr. Levinthal, and the positive reaction of the complement-fixation test obtained by Prof. Bedson, add greatly to the interest of the case.

Addendum by Dr. Levinthal

On May 25th Dr. Johnson sent me a small amount of sputum, stating in a letter that, judging the case from the clinical aspect, he was convinced that it was one of psittacosis. I was able to confirm his

[1] See The Lancet, 1935, i., 1207 and 1226.

clinical diagnosis by the experimental demonstration of the virus.

The sputum, which contained much more mucus and saliva than pus, was emulsified in the tenfold amount of broth by thoroughly shaking with glass beads in a wide-mouth stoppered bottle. The unfiltered material killed one mouse injected intraperitoneally in two days with a pneumococcus of the group IV. The sputum emulsion was filtered through a rather tight sand and paper pulp filter. In view of the fact that the filtrate was nearly clear, it was not filtered further, and was injected intraperitoneally into two mice, one of which was reinoculated with the same amount (1·0 c.cm.) on the two following days. The mice died on the eighth and ninth days with a condition which is characteristic of the infection with a medium amount of virus (cf. THE LANCET, 1935, i., 1209). The virus was abundantly present in the masses of fibrin found in the abdominal cavity, in the perihepatitic and perisplenitic membranes, in the cells of the pericardial serofibrinous exudate, and somewhat less in the spleen. The virulence test, performed with the second subculture in a chicken-embryo medium, revealed the high virulence of the strain with a positive result up to the last titrated dilution of $0{\cdot}5 \times 10^{-9}$ (dead on the 14th day).

It may be added that a budgerigar, which was found dead in the patient's house on June 3rd, had no signs of psittacosis and gave no positive result in the mouse test. On the other hand, the psittacosis virus was detected in one of four budgerigars purchased at random in the middle of June from a bird shop which supplied the patient's aviary.

FRACTURE OF THE RIBS IN WHOOPING-COUGH

BY W. EDGECOMBE, M.D., F.R.C.P. Lond., F.R.C.S. Eng.

HON. PHYSICIAN TO THE HARROGATE AND DISTRICT GENERAL HOSPITAL

FRACTURE of the ribs during a paroxysm of whooping-cough is rare enough to deserve record.

Miss A., aged 18, a well-developed, apparently healthy normal girl, of the non-athletic type, began with a cough on May 24th, 1935. At the end of a week it became clear she was suffering from whooping-cough. On June 7th, during a paroxysm of coughing, she complained of acute pain over the left lower ribs posteriorly. On examination nothing definite was discovered, and the pain was thought to be due to muscular strain. A tight bandage to restrain the movement of the chest wall eased the pain considerably. A week later, on June 13th, during a violent fit of coughing, she felt a severe pain over the lower ribs behind on the right side, and said that she definitely heard a crack as though a bone had broken. On examination there was an area of acute tenderness over the eleventh rib on the right side, and crepitus could be felt on palpation and heard through the stethoscope.

The ribs were tightly bound with strapping which gave considerable relief to the pain. The following day an X ray photograph was taken which disclosed a fracture of the eleventh rib on the *left* side at the junction of the distal and middle third, accounting for the first attack of pain, and a fracture of the eleventh rib at a similar site on the *right* side corresponding to the second attack of pain. Firm strapping made the pain tolerable and the attack of whooping-cough, which was moderately though not excessively severe, ran a normal course of about seven weeks.

Inquiry into her previous history disclosed the fact that during birth she sustained a fracture of the thigh. Labour was prolonged and delivery difficult. At the age of 14 she had a fracture of the right forearm due to a fall in the gymnasium at school. She played the ordinary school games, though not strenuously, and encountered, presumably, the usual rough and tumble knocks without mishap. She has had no illness of any moment.

It was thought desirable to radiograph the bones of the extremities for evidence of deficient calcification. This was done by Dr. C. Hitchcock of Harrogate. The radiograms of the hands compared with those of a normal control of the same age show definitely less density in the bone shadows. The radius and ulna, the tarsal bones, and the tibia and fibula showed similar evidence of deficient calcification. In the epiphyseal ends of the radius at the wrist and to a less extent in the carpal bones and in the lower end of the tibia minute sharply defined points of dense sclerosis about 1 mm. in diameter can be seen. This condition is mentioned by some observers as being associated with fragility of the bones.

The patient has been put on a course of calcium and vitamin D, and it is intended to radiograph the bones again in six months' time.

REVIEWS AND NOTICES OF BOOKS

Diseases of Children

A Work for the Practising Physician. By Dr. M. PFAUNDLER, Professor of Children's Diseases and Director of the Children's Clinic at the University of Munich; and Dr. A. SCHLOSSMANN, Professor of Children's Diseases and Director of the Children's Clinic at the Medical Academy in Düsseldorf. English translation: American Editor-in-Chief, M. G. PETERMAN, Sc.B., A.M., M.D., Professor and Director, Department of Pediatrics, Marquette University Medical School. London: J. B. Lippincott Co. 1935. In five volumes. Pp. 544, 512, 689, 575, and 545. 9 gns. (5 vols.).

THE appearance of a new edition in English of Pfaundler and Schlossmann's standard system of pædiatrics is an event of first importance. Based on the fourth German edition, which appeared just before the death of Dr. Schlossmann, the present English translation has been carried out by 21 American pædiatricians, who have in some places added notes to the original work. This latter is itself widely representative, having been written by over sixty authorities, for the most part German, but including contributors from Austria, Holland, Finland, and America. A valuable feature is the section devoted to tropical diseases in childhood, written by Dr. Wyckerheld Bisdom of Java.

It would be difficult to over-estimate the influence that this work has had on physicians all over the world since the appearance of the first German edition in 1906. It was then largely the work of youthful pædiatricians, but set a standard of scholarship of which the most mature might be proud. With the passage of years, a number of contributors have resigned their association with the work, and others have taken their place; many of the original team, however, including both editors, took part in the production of the fourth edition, and the combination of freshness and maturity is a happy one. In the translation the original has been faithfully rendered into clear, if not always scholarly, English. Unfortunately, however, a few sections fall so far short of the general standard that they seriously mar the

work as a whole; for example, that concerned with diseases of the organs of locomotion is in places almost unintelligible. "Here we deal with phenomena concerning more anatomical than clinical interests" it begins brightly, and continues a shade more solemnly "the heart muscle bellies protrude from the body surface like irregular spindle-shaped tumors. After a duration of many years exitus threatens from an intercurrent infection."

The work is divided into 5 volumes. Vol. I. opens with a history of pædiatrics by Prof. Brüning, which is followed by sections of a more general nature (biology, pathology, and morbidity of early life, examination of sick children, general prophylaxis, child welfare, and therapy), a consideration of nutrition and growth, and a discussion on the special pathology of different age-periods. In Vol. II. deficiency diseases and diseases of the blood, circulatory system, and endocrine glands are included; Vol. III. is devoted to infectious diseases and rheumatism; and Vol. IV. to diseases of the digestive and respiratory systems. Vol. V. comprises the diseases of the genito-urinary and nervous system, and of the motor apparatus, and includes an excellent index. This arrangement necessitates a certain amount of overlapping, but in a work of this nature this is an advantage, since the views of more than one authority are obtainable on different aspects of the same subject. The general production of the volumes is admirable. A heavy surfaced paper has been used, which has made possible the reproduction of radiograms in the text, and the numerous illustrations are well chosen and clear. The selective use of small type has kept the size of the volumes within reasonable limits. As a work of reference there is one great disadvantage to this system of pædiatrics: no exact references to the literature are given, although authors are quoted by name. In spite of this we commend the book as one of the most important and complete systems of pædiatrics obtainable.

Endoscopie et pleurolyse

By O. M. MISTAL, médecin directeur de l'établissement Clovelly, Montana. Paris: Masson et Cie. 1935. Pp. 436. Fr.55

BEGINNING with an anatomical and histological description of adhesions, the author goes on to describe early attempts at their division by open operation. The following chapter on indications and contra-indications of thoracoscopy should be one of the most important in the book if the author had not such an unfortunate craving for completeness that the reader can hardly see the wood for trees. Among the rarer indications for thoracoscopy he gives the confirmation of healing before letting out a pneumothorax; also the observation of the effect of drugs and rays on certain diseases of the lungs and pleura. The thoracoscopic appearances of two forms of syphilis of the pleura are described. The next chapter deals with the instruments used in thoracoscopy; this section contains 80 illustrations, and it is unlikely that any instrument of importance has been omitted. After a description of the anatomy of the pleural cavity, there follow details of the technique of the division of adhesions by cautery, by diathermy, and by a combination of the two. In considering the results of the operation, the writer finds that out of 65 thoracoscopic examinations, adhesions could be divided in 52 cases: 34 patients were improved, 13 stationary, and 5 worse. Perforation of the lung occurred in 3 cases (only 1 during the operation) and hæmorrhage also three times. Serious effusions were noted in 8 cases, slight ones in 34, only 7 cases escaping entirely.

Dr. Mistal describes a new application of thoracoscopy of his own invention. By means of a specially constructed quartz electrode, introduced through a sinus, the pleura may be irradiated with ultra-violet light. In a concluding paragraph he states that "the direct irradiation of the pleural cavity with ultra-violet rays, the introduction of various gases and drugs and of radium, following our own method, will probably have great therapeutic importance in the treatment of certain pulmonary affections." Perhaps they will; but one cannot help feeling that angels would fear to tread some of the paths Dr. Mistal traverses without a backward glance.

Heart Disease in the Tropics

By H. O. GUNEWARDENE, M.B., B.S.Lond., D.M.R.E. Camb., late Clinical Assistant, National Heart Hospital, London; Consulting Cardiologist, Columbo, Ceylon. India: Butterworth and Co., Ltd. 1935. Pp. 101. 8*s*. 6*d*.

THIS book should interest others besides those whose work is in the tropics. Dr. Gunewardene believes that new lines of investigation may suggest themselves to those interested in heart disease from reading a description of, for example, such a condition as rheumatic heart disease as it manifests itself in an entirely different climate and setting. One of the most interesting chapters deals with cardiac disability as a result of ankylostoma infection which, according to the author, is comparable with rheumatic fever as a common cause of heart disease in the tropics. While studying this condition he was greatly impressed by the variety of murmurs which might develop during the course of the disease, and he was able to convince himself and others that not only systolic but also diastolic and presystolic murmurs might be heard, disappearing as the patient recovered. To illustrate these observations a full account of several cases is given. Apart from this a review is given of cardiovascular phenomena in tropical climates, and sections on rheumatic heart disease, diabetes, diphtheria, syphilis, and high blood pressure. The value of digitalis, oxygen, and potassium iodide is briefly discussed, and a short section on the effect of altitude on cardiovascular cases is included.

Inhalation Therapy Technique

By W. E. COLLISON. With Foreword by Sir BRUCE BRUCE-PORTER, K.B.E., C.M.G. London: William Heinemann Ltd. 1935. Pp. 76. 5*s*.

ALTHOUGH beginning with a historical introduction describing early types of atomiser, this book is really an advertisement for the author's Apneu inhaler. This fact does not detract either from the well-known merits of the instrument described, or from the value of the information given. Major Collison's inhaler is mounted on an oxygen cylinder from which the gas can be made to pass through medicated solutions; the high pressure of the oxygen jet results in vaporisation into smaller particles than can be obtained by atomisers working with low pressures. The oxygen with its content of drugs in fine suspension is administered to the patient by means of a rubber bag and face mask, similar to those used for nitrous oxide anæsthesia. The apparatus is simple to use as it only requires a turn of the handle to give one or other of the atomised drugs or a mixture of the two. With another setting pure oxygen can be given.

After a clear description of the apparatus the writer proceeds to give some recommendations for its use in,

various affections of the respiratory tract. The preparations advised are given under such trade names as apneugene and campheugene, but the composition of each is given. Asthma, bronchitis and emphysema, bronchiectasis, pulmonary tuberculosis, laryngitis, and even the common cold has its appropriate inhalation. The last part of the book consists in extracts from articles in medical publications in which the apneu method has received favourable mention.

The medical profession has, undoubtedly, been backward in inhalation therapy, and even the ridiculously inefficient administration of oxygen through a glass funnel has not been altogether given up; in Major Collison's book readers will find useful information about an efficient method of inhalation.

What of the Child?

By ANDREW KEFALAS, M.A., M.B., Ch.B. London: William Heinemann Ltd. Pp. 187. 5s.

IN the flood of books on children this little volume takes a useful place. The author describes the psychophysics of development and peculiar habits. He discusses the rôle of the parents in upbringing, in discipline, and in punishment. He describes the play activities of children, their daydreams, their fears and peculiarities of character. He also gives special attention to the important problem of puberty and adolescence, and the various social and personal anomalies which colour youthful development. He also has some sensible advice to give on sex instruction. The book as a whole is intelligent and sympathetic, and can be recommended not only to parents but to general practitioners who are awakening to the psychological aspects of child development.

Introduction to Pharmacology and Therapeutics

Fourth edition. By J. A. GUNN, M.A., M.D., D.Sc., F.R.C.P., Professor of Pharmacology in the University of Oxford and Fellow of Balliol College. London: Humphrey Milford, Oxford University Press. Pp. 237. 5s.

THE fact that this handbook has reached its fourth edition within five years is a clear indication that there can be few students and teachers of medicine who are not aware of Prof. Gunn's valuable work. The book is intended to be read before the student attends lectures on pharmacology; but it provides such a comprehensive survey of the whole subject that it might well be read by anyone desiring to refresh his memory as to the principles underlying the art of healing. The new edition shows few changes, for little more than a year has elapsed since the third edition was published, and it can be warmly recommended as a sane, critical, and attractive account of the subject.

Electrocardiography

By CHAUNCEY C. MAHER, B.S., M.D., Assistant Professor of Medicine, North-Western University and the Montgomery Ward Medical Clinics. London: Baillière, Tindall and Cox. Pp. 250. 18s.

Dr. Maher's book has been written not for the cardiologist but for practitioners and for those who specialise in departments of medicine other than cardiology. Its aim is to enable them to correlate the electrocardiographic with the clinical findings. The two opening chapters deal with the clinical classification of heart disease and the arrhythmias, while in a short third chapter the author outlines the anatomy of the conducting system. Chapters 4 and 5 are devoted to technique and to a consideration of the features of normal records. From here onwards the author follows a uniform scheme. Each disorder of the heart's mechanism is illustrated first, by a drawing of the Purkinjé system to indicate the site of the lesion, secondly, by a diagrammatic electrocardiogram, and thirdly, by actual records. To each record is appended a tabulated analysis, the first part of which deals with the clinical and the second with the electrocardiographic diagnosis. This method makes it easy for the novice to interpret the records, but provides somewhat tedious reading for those readers who have some previous knowledge of the subject.

The chapter which appealed to us most was that dealing with axis deviation. This subject is illustrated by some excellent radiograms which enable the reader to compare the anatomical with the electrical axis of the heart in cases where the brunt of the lesion falls on the right and left ventricle respectively. It also illustrates well the range of normal variation in the electrical deviation associated with differences in the lie of the heart. The records are well chosen and clearly reproduced.

Encyclopedia of Veterinary Medicine, Surgery and Obstetrics

Second edition. By GEORGE H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A., F.R. San. I. Professor of Medicine and Hygiene, Royal Veterinary College, London; Hon. Consulting Veterinary Surgeon to the Zoological Society, London. London: Humphrey Milford, Oxford University Press. In two vols. Pp. 1652. 6 gns.

THIS encyclopædia has been brought up to date, and some chapters, including those on actinomycosis, actinobacillosis, milk fever and braxy, have been rewritten. New chapters are supplied on lamb dysentery, swine influenza, spinal anæsthesia, surgical narcosis with avertin and nembutal, gland grafting, infection and immunity, and a veterinary pharmacopœia has been added. As regards arrangement, the first volume of 836 pages contains a section dealing with diseases caused by bacteria or filtrable viruses, the conditions being grouped according to the animal species; this system is not, however, adhered to, since the chapter in which contagious mastitis is discussed is to be found amongst surgical conditions. There follow sections which discuss protozoal, constitutional and local diseases. The second volume of 816 pages deals with surgery and obstetrics. Most of the chapters have been written by Prof. Wooldridge, but 48 other workers have contributed articles on subjects with which they are particularly familiar. Chapters on horse sickness, contagious bovine pleuropneumonia, bluetongue, swine fever, piroplasmosis, acariasis and mastitis deserve special mention; whilst the veterinary pharmacopœia and the chapter on infection and immunity are useful adjuncts to the new edition. Some aspects of veterinary medicine have received inadequate treatment: thus the whole subject of plant poisons is dealt with in only six pages, whilst with two or three exceptions all diseases of poultry are omitted.

In collecting and assembling a mass of scattered and unavailable information, Prof. Wooldridge has done a great service to the cause of animal health; and if this encyclopædia is amplified in certain places in future editions, the veterinary professor will find it almost indispensable.

THE LANCET

LONDON: SATURDAY, AUGUST 17, 1935

SPECIFIC CONTROL OF WHOOPING-COUGH

LIKE measles, whooping-cough is not generally notifiable, and our idea of its incidence must be based largely on estimates such as those of STOCKS and KARN, quoted by Drs. PATERSON, BAILEY, and WALLER in the paper which appears in our present issue. Our knowledge of the morbidity due to its complications is even less exact. The mortality, owing to the enduring nature of the illness, is probably understated to a lesser degree than that of measles, in which the deaths tend to be ascribed to the complications to the exclusion of the primary infection; indeed, the figures given by Dr. STOCKS in his paper on p. 386 suggest that whooping-cough is sometimes blamed for deaths which are chiefly the result of influenza epidemics. But the fact remains that, though the public still looks on it lightly, this disease is now the most formidable of the acute specific infections of childhood.

The therapy is notoriously unsatisfactory. The new drugs proposed are legion. Vaunted for a time they are finally rejected in favour of the few well-tried but not always reliable sedatives prescribed by our predecessors. Although success for vaccine therapy has been claimed by some, most observers report results which are at best equivocal. Serum treatment is little more promising: Dr. PATERSON and his collaborators note that JUNDELL seems to have secured some amelioration of the attack by means of the injection of reactivated adult-immune serum, but they themselves are unable to record any apparent improvement from the treatment of a series of cases with convalescent serum. Specific prophylaxis on the other hand arouses greater hope. The work of MADSEN in Denmark and of SAUER in the United States of America is particularly well known, and opinion, if not unanimous, is much less divided over the value of vaccines in prophylaxis, provided they are prepared from fresh strains in the correct antigenic phase and injected in adequate doses at a sufficient interval before exposure. In recording the results of prophylactic injection of convalescent serum PATERSON, BAILEY, and WALLER are rightly cautious in expressing an opinion about its value. Their series of 95 contacts with 55 controls is too small to have statistical significance, but the fact that 42 per cent. of the presumably infected contacts did not develop the disease, in comparison with only 11 per cent. of the controls, is a basis at least for further investigation, and this no doubt will be made.

In an attempt to determine whether a child had had an infection with the Bordet-Gengou bacillus on some previous occasion or whether vaccine prophylaxis in a non-infected child had been of any avail, Dr. PATERSON and his co-workers have made use of a skin-test in four groups of children. An area of hyperæmia, about three-quarters of an inch across, appearing 24–48 hours after the intradermal injection of a minimal amount of vaccine was considered as positive and an indication of an allergic state following natural infection with, or vaccine prophylaxis against, the Bordet-Gengou bacillus. The utility of such a test, if its reliability can be confirmed, is obvious. The results recorded are peculiarly interesting, inasmuch as SIEBLER and OKRENT[1] employing a similar test considered that hyperæmia indicated susceptibility and a negative reading immunity following vaccine prophylaxis—an interpretation exactly opposite to that of the present observers.

INDUSTRIAL MEDICINE IN FLUX

THE organisation of industry is undergoing changes and developments that are both rapid and fundamental. In the course of comparatively few years it has had to cope with war, boom, depression, and a constant flow of new inventions. It is in a state of flux and he would be rash who would attempt to forecast the form of the next phase of stability. In the midst of this vital but restless period the study and practice of industrial hygiene and medicine have progressed if not in a spectacular yet in a very encouraging way. It has been impossible for the medical needs of industry to reach the stage of clear definition while industry itself is undergoing a phase of extensive internal change, but in spite of the fact that industrial medicine is still adolescent great strides have been taken. A stage has been reached where, given intelligent coöperation, the medical hazards of occupation can be countered and work made reasonably safe and congenial. It is useless to deny that this state of affairs is made easier of attainment where the unit of production is large, and whatever drawbacks there may be to so-called rationalisation ease and efficiency of medical supervision is not one of them. Centralised control of production is a powerful ally of industrial medicine. How far this tendency will spread is uncertain; meanwhile the vast majority of industrial concerns employ less than a hundred workers, and the problem of the medical supervision of these remains. A medical scheme does not work economically in units of less than a thousand workers and the alternative method of groups of small factories coöperating for medical services has unfortunately received practically no trial. It is something well worthy of experiment.

Many matters which call for medical supervision at work are not receiving, or are not capable of receiving, attention in any other way. These range from the proper selection of staff and the prevention and treatment of hazards and accidents to the equally important coöperation with private

[1] Siebler, K., and Okrent, S.: Jour. of Pediat., 1934, iv., 188; see THE LANCET, 1934, ii., 877.

doctors over such things as return to work combined with treatment, and consultation over individual traits and circumstances that only become manifest in the patient's working environment. Our columns in the past few years have contained numerous contributions dealing with these matters, and future developments were considered at the recent congress of the Royal Sanitary Institute in Bournemouth, when a section of hygiene in industry collaborated with the Industrial Welfare Society. At this congress Dr. T. E. A. STOWELL emphasised the foundations common to all medical services in industry; no matter what the needs of a special concern might be there is an A.B.C. of industrial hygiene without which nothing can succeed. Dr. W. D. JENKINS outlined the opportunities and duties that exist for employers to help and care for their older workmen. Important now this is a side that will gain in importance if the average age of the population continues to rise and the proportion of older people to increase. Even after they have left industry these older people can be useful citizens and it is a social duty to see that in their later years their work does not unnecessarily impair their powers of happy and useful life. The distribution of goods is undergoing rapid changes and not the least of these is that more people are being employed in what may be called the ancillary services of production. Though not strictly industrial the work of the sales-shop and store is organised on lines which call for medical supervision difficult to apply except in large single establishments. Dr. M. L. DOBBIE-BATEMAN dealt with the shop assistant and the meeting was notable for the emphasis laid on the medical problems of retail selling. Working with a large retail staff, albeit under the ideal condition of having them almost under one roof, Dr. DOBBIE-BATEMAN has utilised her opportunities to collect information about an occupation which in general is scattered in small units but which nevertheless calls for as much medical attention as the ordinary run of factory work. Lord TRENT, who presided over the section, was able to draw on his own administrative experience and stated frankly that the picture of the life of a shop-assistant in big cities can be an unpleasant one unless the employer is expertly advised and takes medical considerations into account in a fair and sympathetic manner.

The boundaries of occupational medicine are thus widening from mines and factories to offices and shops—from mere hazards to the deeper, more intimate and individual psychological maladjustments of the worker. That this extension could under improper administration result in victimisation is obvious, but the phase of suspicion is dying down as industrial medical officers continue to prove that they are doctors to their patients before they are servants to anyone, and they are gradually widening their own function to embrace those wider economic interests which are so potent in the making or breaking of the health of individual workers. Lord TRENT showed a deep understanding of this social aspect of medicine when he stated that "the removal of that perpetual fear of losing one's job which besets working people of all ages [is] the greatest human problem that will have to be solved in the course of this century." The acknowledgment that economics and medicine are inextricably interwoven is at once the justification for industrial medicine and its greatest urge to progressive and far-seeing development. The medical profession as a whole has a deep concern in the trends of industrial and economic organisation because while a movement, such as for instance large-scale grouping, may bring forward new problems it also produces new opportunities; and who will deny that in a complex world centralisation is essential if we would remain sane and that through coöperative effort preventive medicine is made easier and treatment more comprehensive and efficient? Industrial medicine will develop not with the object of increasing sectional profits but because an industrial state cannot afford to leave untilled a most fruitful and economically efficient field of preventive and curative medicine.

TRAINING THE SQUINTING EYE

THE interest now taken in the treatment of squint, whether with or without operation, shows no signs of abating. Miss DOBSON'S most recent contribution to the subject [1] will be found of great practical use. She describes a device for the use of children whose concomitant squint has already been overcome, or nearly so, either by the wearing of glasses or by other methods, but in whom one eye remains to a greater or less extent amblyopic from disuse. Suppression of the image in a squinting eye is the great obstacle to successful orthoptic treatment. Hitherto complete occlusion of the non-squinting eye has been the favourite method for developing vision in a squinting one, and in the case of young children with constant monolateral squint it is often the only practicable one. The ideal method of occlusion, which admits of some indirect vision on the temporal side of the field but completely shuts off direct vision, was set out in our columns (July 27th, p. 183) by Mrs. THORNETT-JOHNSON, but she admits that for any child after the age of six with vision in the squinting eye reduced to much below 6/60 it is a very trying and long treatment. For older children, although it may be possible, unless the vision of the squinting eye has improved considerably beyond 6/60 it must necessarily be a serious handicap to education. In Mrs. THORNETT-JOHNSON'S experience the average time taken at a clinic to restore the vision of a squinting eye seeing less than 6/60 to normal has been 18 months, the shortest being 6 months and the longest 3 years. Any method of quickening the pace would be welcome. When the child is able to read, when the squint is only slight or occasional, and when the vision of the squinting eye is good enough, with refraction

[1] The Amblyopia Reader: A System of Eye-sight Development. With foreword and notes by Margaret Dobson, M.D. London: Rembrandt Photogravure Ltd. Pp. 93. 15s.

corrected, to read large print by itself, it is easier to overcome the remaining amblyopia by employing it in harness with the good eye; and in this way there is then not only the urge to interpret the image that falls upon the retina of the squinting eye but also the stimulus of binocular vision.

Many varieties of apparatus are in vogue to effect this object. One of the most successful is the cheiroscope (or eye awakener) of Maddox. Various forms of "amblyoscope" and "kinetic stereoscope" and the complicated (and necessarily expensive) "synoptophore" are in use for this purpose. There is room for a simpler and less expensive method of effecting the required object and one which may be used at home. Miss DOBSON'S method is ingenious. She has had a number of nursery rhymes and simple stories printed with certain words and pictures half in black and half in orange. The vision of the two eyes of the child having been made approximately equal, if necessary by means of a Chavasse glass before the good eye, a ruby red shield is also placed before that eye so that the halves of the words or pictures printed in orange are completely obliterated, but so that the amblyopic eye with its refraction corrected can see the whole of each word, the size of the print being graded according to the degree of the amblyopia. In this way the help of the good eye is called in to assist in the development of the sight of the bad one. The idea is a good one and will, we anticipate, be extensively used. Anyone proposing to undertake the non-operative treatment of squint in children would do well to inspect the methods in use at existing clinics before purchasing expensive apparatus. Apart from apparatus a large amount of both patience and tact are required for success.

CLEARING THE TUBES IN ASTHMA

IN a large proportion of patients with asthma the respiratory tract is unhealthy. The nasal mucosa is swollen, the ostia of the sinuses are blocked, and bacteria flourish in the stagnant secretions. There is a persistent low-grade bronchitis and the mucous plugs expectorated from the smallest bronchioles contain leucocytes and bacteria, the latter often in almost pure culture. Acute episodes such as sinusitis, hæmoptysis, pleurisy, and pneumonia are abnormally common. The pulmonary lesions, like those in the nose, must often be secondary to hypersecretion of mucus and œdema of the epithelium, which block the small bronchioles and favour stagnation and infection. Exceptionally, the secretion of mucus may be so great as to obstruct the whole bronchial tree and cause death from asphyxia, as in a case reported by BRULÉ and others.[1] A less uncommon accident is massive collapse from obstruction of a single main bronchus. In theory, the unhealthy state of the respiratory tract may be regarded as a result of asthma, but in practice it is known to be an important factor in the perpetuation of the disease. Methods to restore a healthier state of the respiratory system are therefore an important part in the treatment of asthma, and in this country the Asthma Research Council has emphasised the value of respiratory exercises which empty the bronchi and deflate the lungs. SHAMBAUGH and ALTER[2] in America report good results in fifty cases of bronchial asthma from a regimen based on elimination by postural drainage of the accompanying bronchial and pulmonary exudate. The patient kneels on a chair or stool with his hands on the floor, and coughs as nearly continuously as possible, until the bronchi have been completely emptied. The treatment is carried out twice a day, for a minimum of three minutes, and any coughing between is regarded as a sign that insufficient evacuation has been accomplished. A simple saline mixture given before each treatment may loosen the secretion and render its evacuation easier. The manœuvre is reminiscent of the late Dr. TUCKER WISE'S postural method[3] of treating phthisis designed to promote drainage of the bronchial tree. He encouraged his patients to lie for half an hour three times a day on an inclined plane with the head lower than the hips, and the gratifying results he obtained were no doubt due to the relief afforded by the maintenance of a free airway.

It is rather unusual to give treatment by inoculation to young children in this country, but HAIBE,[4] director of the Bacteriological Institute at Namur, considers it very important to treat asthma vigorously as early as possible before permanent damage has been done. He claims a 90 per cent. recovery-rate in young children with asthma after treatment by the application of antivirus to the nose, and the subcutaneous injection of autogenous vaccines made from sputum. The antivirus was a stock preparation made from a variety of organisms isolated from patients with asthma. When the patient was too young to bring up sputum, material for the preparation of the vaccine was made from swabs from the larynx. In this country we may not be so sanguine about the action of antivirus as our French contemporaries, but in any effort to clear the respiratory system the nasal passages should not be neglected, and the use of nasal sprays or the displacement technique of PROETZ should be remembered.

[1] Brulé, M., Hillemand, P., Delarue, J., and Netter, A.: Bull. et mém. Soc. méd. hôp. de Paris, June 10th, 1935, p. 981.

[2] Shambaugh, N. F., and Alter, S. M.: Science, Feb. 22nd, 1935, p. 210.

[3] THE LANCET, 1908, i., 1546.

[4] Haibe, A.: Scalpel, July 20th, 1935, p. 889.

THE Medical Research Council have appointed Prof. J. A. Nixon, C.M.G., M.D., F.R.C.P., and Dr. R. E. Lane, M.R.C.P., to fill vacancies in the membership of their Industrial Health Research Board.

INDEX TO "THE LANCET," VOL. I., 1935

THE Index and Title-page of Vol. I., 1935, which was completed with the issue of June 29th, is now ready. A copy will be sent gratis to subscribers on receipt of a post card addressed to the Manager of THE LANCET, 7, Adam-street, Adelphi, W.C.2. Subscribers who have not already indicated their desire to receive Indexes regularly as published should do so now.

ANNOTATIONS

LIFE WITHOUT BREATHING

Singh reports [1] that a dog has been kept alive for 16 minutes without breathing oxygen through the lungs; it was supplied with oxygen intravenously while under an increased atmospheric pressure amounting to three atmospheres. This crucial experiment was ended abruptly by pulmonary embolism, the result of a fault in the injecting apparatus which allowed a sudden gush of oxygen into the veins. Otherwise there seems no reason why it should not have continued much longer; and herein may lie the germ of an important advance in oxygen therapy. Previous workers have injected oxygen into the veins, but in human subjects there is known to be a grave risk of pulmonary embolism if the rate of injection exceeds about 0·75 c.cm. per kg. of body-weight per minute. While it has been suggested that small quantities given at intervals might provide useful means of oxygen administration, this method has not so far been scientifically justifiable in the treatment of anoxæmia. Singh has investigated its possibilities in two stages. First he determined the dose of oxygen that could be absorbed intravenously under ordinary atmospheric pressure. He found that cats could absorb about 5 c.cm. of oxygen injected in about ten minutes, and that other animals of different species could take larger doses in relation to their weight. The amount of gas absorbed depends on various factors including the distance between the point of injection and the heart and lungs, and the calibre of the veins along which the bubbles travel. But it was clear from the experiments that, owing to the limitations imposed by the calibre of the available veins and the rate at which the oxygen can be carried away, injection at ordinary atmospheric pressure can supply only a comparatively small proportion of an animal's oxygen requirements. Singh therefore proceeded to repeat his observations within the pressure chamber at the premises of Siebe, Gorman and Co. At an increase of one atmosphere it proved possible to inject intravenously 30–40 per cent. of the oxygen required by a cat without any harmful effects, and at two atmospheres about two-thirds of the oxygen required by a dog was introduced. The crucial experiment, as already stated, showed that at a pressure of three atmospheres the whole of a dog's oxygen requirements could be met by intravenous injection. It was, of course, necessary for the complete experiment to be performed in the pressure chamber, and the operator had himself to work there. During the injection the dog's respiration was almost normal; a soda-lime tube attached to the trachea, and leading to a rubber bag absorbed the expired carbon dioxide.

The theoretical likelihood that under positive pressure enough oxygen can be injected to meet all the needs of an animal has thus been justified in fact. The application of this finding to problems of chest surgery, resuscitation, and the relief of anoxæmia is the next, and obviously very difficult, step. An alternative route for the supply of oxygen when large areas of normal aerating surfaces of the lungs are put out of action by, for example, a diffuse broncho-pneumonia would get over difficulties at present insurmountable (except in milder cases where the oxygen-tent is very useful). Injection of the gas, if it proves practicable, should be a valuable means of coping with respiratory failure of this type.

[1] Singh, I.: Jour. of Physiol., 1935, lxxviv., 315.

AIRCRAFT AND YELLOW FEVER

THE risk of yellow fever spreading to parts of the world at present immune from this disease has been much increased in recent years by the development of aerial transport, and public health authorities are everywhere becoming alive to the danger. One side to the problem of diminishing this risk consists in preventing the transport of the mosquito vector, *Aedes ægypti*. In a communication in the monthly bulletin of the Office International d'Hygiène Publique for July, Dr. C. L. Williams and Dr. W. C. Dreesson, of the United States public health service, confine themselves to this aspect of the subject. They dismiss as impracticable the frequent fumigation of aircraft with hydrogen cyanide; fumigation with "carboxide" (ethylene oxide and carbon dioxide) they find not to be satisfactory on account of the great weight of necessary apparatus and the high concentration of this gas required to kill mosquitoes; and they finally recommend a concentrated extract of pyrethrum (2 per cent. of pyrethrin in light oil) distributed by an atomiser in a very fine cloud at the rate of 2 to 4 grammes per 1000 cubic feet. That such a method, applied with care and conscience, should destroy all the mosquitoes on board there is no reason to doubt. But that such a method should be necessary at all is a sorry reflection on the state of the sanitary organisation at the aerodromes in question. To free the native quarter of a city from *Aedes ægypti*, or to control it completely in a seaport full of small craft and lighters, is admittedly not an easy matter. But there is no reason whatever why a well-planned aerodrome with an efficient sanitary gang should produce a single specimen of this purely domestic mosquito.

AVOIDANCE OF REACTIONS AND FAILURES IN DIPHTHERIA IMMUNISATION

ACTIVE immunisation against diphtheria, when carried out in residential institutions, implies, or should imply, preliminary Schick tests. In clinics, however, practical considerations may compel the omission of the test or impose a single reading which is usually made a week after its performance. In either event the medical officer is deprived of the evidence of protein sensitivity afforded by the appearance of the transient pseudo-element. A pseudo-positive reactor to the test is likely to be a reactor to the prophylactic, the dosage of which may be modified accordingly. To provide an indicator of sensitivity to protein contained in the prophylactic the Moloney test, the *intradermal* injection of a minute dose of diluted toxoid, has been used upon a large scale in Canada and advocated in this country. Dr. Chesney [1] considers that this test, although desirable, is a troublesome procedure in practice; and it is one which, it may be added, Swyer [2] found to be not an accurate index either of the liability to, or severity of, the reactions to be expected after the injection of formol-toxoid in the case of young adults. Further evidence as to the reliability of the test in children will doubtless be forthcoming, but Chesney prefers to employ what he terms the "detector dose," a preliminary dose of formol-toxoid injected *subcutaneously*. Several batches of toxoid of varying but high antigenic value (but of different origin and dosage to

[1] Chesney, G.: Ann. Report M.O.H. Borough of Poole, 1934, p. 15.

[2] Swyer, R.: THE LANCET, July 6th, 1935, p. 22.

the toxoid used by Swyer) were employed by Chesney in the immunisation of over 700 children. For children up to fourteen years of age a preliminary detector dose of 0·2 c.cm. of toxoid was injected; this was reduced to 0·1 c.cm. for those over fourteen. Usually no local redness or tenderness followed the injection of 0·2 c.cm. of formol toxoid; occasionally an area of redness 1–2 cm. across resulted and rarely one of 2–5 cm. After the detector dose, which "contains 200 times the amount of toxoid used for the Moloney test and in itself acts as a primary stimulus," two doses of the prophylactic were injected subcutaneously at fortnightly intervals. Non-reactors received, presumably, two full doses of 1 c.cm. of toxoid; slight reactors, 0·4 c.cm. and 0·6 c.cm.; but the particularly sensitive were injected with toxoid-antitoxin floccules (T.A.F.). Among a group of 332 children only 10 reacted to the detector dose to such a degree as to make it desirable to change to this preparation, which is of high antigenic value and causes but few and slight reactions even in adults.

Chesney now uses a formol-toxoid of 58 Lf. per c.cm. in a routine dosage of 0·6 c.cm. and 1 c.cm. at fortnightly intervals following the detector dose. He states that the number of Lf. units in this course of injections is "in excess of that contained in three 1 c.cm. doses of the formol-toxoid generally used in this country." Reaction to the dose of 0·6 c.cm. is followed by a dose of the same magnitude instead of the full final dose of 1 c.cm. The proportion of Schick-immunes produced by this method and this prophylactic appears to be very satisfactory, immunity to Schick-negative level being attained in more than 90 per cent. in a few weeks. Chesney thinks that the occurrence of clinical diphtheria, caused by the gravis strain or a heavy infection with a milder strain in a few persons who had previously been immunised and found to be Schick-immune to the ordinary test, suggests that the level of immunity thus indicated may not be sufficiently high. He has therefore made some observations with a more potent Schick toxin supplied by Dr. R. A. O'Brien. This preparation, although containing no more free toxin than the standard fluid, had four times its combining power, owing to the presence of "toxoided" toxin. Tests with the two preparations were made simultaneously upon a group of 45 children who had received a course of formol-toxoid. In 5 cases (11·1 per cent.) the readings were positive to the fourfold test: of these, 4 were negative to the standard test and 1 was positive. The remaining 40 children were negative to both tests. From the results in this small series Chesney draws the rather sweeping conclusion that "in every hundred children in his area treated with the prophylactic course, there are two or three who fail to develop immunity of the level indicated by the standard Schick [test] and a further nine who are not satisfactorily immune in spite of their negative reading to the standard Schick test, and that this 9 per cent. are the Schick negative children whose 'Schick immunity' will break down before a heavy or markedly virulent infection."

It would have been interesting and possibly instructive if this group of 5 children had been tested again with the fourfold Schick toxin a week later. It may be suggested that some at least who were at first positive to the fourfold test would, as the result of the secondary stimulus thereby provided, have become negative reactors. Whether the secondary stimulus of Glenny be Schick toxin or the diphtheria bacillus itself, its effect is rapidly to raise the antitoxic titre of the blood.

SYPHILITIC AORTITIS

It has been suggested in Germany that syphilitic aortitis is alarmingly on the increase, and Dr. C. Müller,[1] of Oslo, has been studying the post-mortem records of the Ullevaal communal hospital to see whether the German conclusions are applicable to Norway. His material covers the period 1905–33, and consists of 14,258 autopsies, at 455 of which syphilitic aortitis was found (3·2 per cent. of all the examinations). Though there was a large increase in the annual number of such cases during the 27-year period under review, this is fully accounted for by the increase in population, and Dr. Müller suspects that the German findings merely reflect a change of fashion in diagnosis and nomenclature. Of 244 Norwegian cases of syphilitic aortitis diagnosed as such post mortem between 1920 and 1931, about two-thirds were complicated by such conditions as aortic insufficiency and aneurysm. The correct diagnosis was made clinically in only 36 per cent., the complicated cases being recognised in 51 per cent., and the uncomplicated in only 6 per cent. These are figures disconcerting to even the most hardened clinician, and Müller indulges in calculations showing how many of the diagnostic oversights could have been avoided by systematic examination of the patient. In only 50 of the 244 cases had an attempt been made to obtain a history of syphilis; and only in 62 per cent. had the Wassermann reaction been tested. Other serological tests might also have been exploited more fully, although some 20 per cent. of the cases under review proved to be sero-negative. Müller also pleads for the more general and regular use of X rays, and a more comprehensive study of the clinical picture. It is not enough to rely on a diastolic murmur over the heart, for this was observed in only two-thirds of his cases of aortic insufficiency; a systolic murmur is, in his experience, at least as common as a diastolic murmur in such cases. The patients with neurosyphilis in his post-mortem material died comparatively early, presumably because syphilis of the nervous system runs a more rapid course than syphilis of the aorta, the prognosis of which is not altogether desperate. Indeed, less than half of the Ullevaal patients suffering from syphilitic aortitis died of disease of the heart or aorta. The average age at death was between 56 and 57, and over 12 per cent. of all the patients were more than 71 years old. There was even a handful of octogenarians among them. Aneurysms proved less fatal than might have been anticipated, for only 19 per cent. of their owners died from rupture of the aneurysm or pressure exerted by it on the surrounding structures.

SHIVERING

Some interesting experiments on shivering are reported[2] from the National Hospital, Queen-square, comprising observations both on normal persons and on patients with selected nervous lesions. The condition was produced by immersing the legs first in a warm and then in a cold bath, rectal and skin temperatures being recorded meanwhile from thermocouples suitably placed. The thermocouple in the rectum was 5 cm. from the sphincter, and almost certainly gave the blood temperature. This fluctuated surprisingly, rising by about ½° during the warm bath and falling about 1½° during the cold. In the normal subjects shivering started shortly after the change of bath and stopped after a change in the reverse direction.

[1] Norsk mag. f. laegevidensk., July, 1935, p. 673.
[2] Uprus, V., Gaylor, G. B., and Carmichael, E. A.: Brain, 1935, lviii., 220.

Although the long latent period—three minutes or so in each case—was against a reflex originating in the skin, there was nothing definite in the records to exclude this. Their most striking feature, nevertheless, was the close association of onset and cessation of shivering with the peak and trough of blood temperature respectively, suggesting that a change in this temperature was the effective agent. This theory was confirmed by the case of the paraplegic patient who showed similar changes in blood temperature and similar shivering reactions, but in whom reflex effects were excluded by the fact of the stimulus being confined to the legs, innervated from below the lesion, and the shivering to the upper parts of the body, innervated from above the lesion. Afferent stimuli were further eliminated by the observation that a patient with a lesion of one thalamus and corresponding loss of sensation from the opposite half of the body responded in the same way as a normal person. There is therefore little doubt that shivering, or its cessation, is the result of a change in blood temperature, although the actual level at which this takes place seems to be of minor importance. The efferent path of the motor impulses must start from a localised part of the central nervous system, for if the change in blood temperature acted on all the spinal segments shivering would take place below the level of a transverse lesion—which, as we have seen, does not happen. The origin of the impulses has not been located, but there is evidence that they are transmitted by the extrapyramidal tracts of the spinal cord, shivering having been found absent in one patient below the level of an operative section of the spinothalamic tracts; the actual spinothalamic lesion could not produce this effect, there was no evidence of involvement of the pyramidal tracts, and other cases (hemiplegic) had shown that shivering could occur, apparently normally, after considerable damage to them. This impression as to the extrapyramidal nature of the impulses was strengthened by the finding that damage to the cerebellum had a distinct influence on shivering, converting the ordinary clonic movements into choreiform movements. The investigators point out that shivering occurs from other causes, such as emotion and fever, and we hope they will extend their studies to these conditions.

ADHESIVE STRAPPING AND THE PATCH-TEST

IT is unfortunate that so useful a preparation as adhesive strapping is often the cause of an irritation dermatitis; and as a rule the more efficient the strapping the greater the risk of dermatitis, elastic adhesive strappings sometimes giving an incidence as high as 25 per cent. Much of this dermatitis is mechanical in origin, arising from the tackiness of the application, and is comparable with the eruptions provoked in bakers by the sticking of dough to the skin. This type of dermatitis from adhesive plaster is not serious and readily subsides under suitable and simple treatment. A few cases, however, are traceable to an allergic sensitiveness towards one or other of the ingredients of the plaster, and this is more awkward, since limited contact may give rise to acute and distressing lesions which may persist for weeks. Schwartz and Peck [1] have investigated this problem by patch-testing over a hundred employees engaged in the manufacture of adhesive tape. They find that the resins employed are common causes of allergic dermatitis—all of 13 cases showing the condition were resin-sensitive—and that rubber is an occasional cause. This has been recognised by manufacturers, but efforts to find a non-irritating resin have not yet proved successful. In employing adhesive strapping for ordinary patch-tests one must bear in mind the possibility that the strapping itself may cause irritation and so obscure the results.

Elastic adhesive plasters probably find their greatest use in treatment of gravitational affections of the legs, but recent writings suggest a tendency to abandon the valuable technique evolved by Dickson Wright,[2] partly because dermatitis is a common sequel. This hazard can usually be avoided however by some simple expedient such as the application of the bandage over a protective covering like lint, gauze, cotton stockings, or zinc-gelatin paste. Another simple method, which can always be adopted in making patch-tests and is often suitable for legs, is the application of the elastic bandage reversed so that it does not adhere to the skin but to itself.[3]

The patch-test is now a routine examination for specific skin sensitiveness but not all workers subscribe to the same technique, and it seems desirable that standard conditions should be observed to allow a fair comparison of results. Enough work has been done to show that patients actually suffering from a sensitisation dermatitis will react immediately to a patch-test with the causative agent. Those who are not actually suffering from a dermatitis but are being tested for possible sensitiveness may show a reaction delayed not only for 24–48 hours, as often supposed, but for as long as 14 or even 24 days.[4 5] This means that observation should be continued at least a month after routine investigations such as those described by Schwartz and Peck. Further, the patch-test is meant to demonstrate allergic sensitiveness and only a limited contact should be necessary to provoke such a reaction. The usual 24-hour application is if anything on the generous side, and has indeed been known to produce a simple traumatic reaction which tends to confuse the issue. It should certainly not be necessary, and seems undesirable, to leave patch-tests in place for 48 hours as suggested by these writers and by others recently.[6] For the same reason it is questionable whether a covering of gutta-percha or oil-silk over the patch-test is an advantage.

MALARIA AND HOOKWORM

As one of the lessening number of those who saw the early stages in the fight for the prevention of malaria, Sir Malcolm Watson has put on record [7] something of what has come within his own knowledge and experience during a time when, so he puts it, the spirit of high adventure filled the veins of medical men of many races. The opening of the story is taken up by an account of his noted attack on malaria at Klang and Port Swettenham in 1901, the fight seeming to be almost without hope in view of the high temperature, the rainfall of 100 inches a year, and the amount of swamp and tropical vegetation round them. So much was this the case that the High Commissioner

[1] Schwartz, L., and Peck, S. M.: Pub. Health Rep., U.S.A., June 14th, 1935, p. 811.

[2] Wright, A. D.: THE LANCET, 1931, i., 457.
[3] Ingram, J. T.: Brit. Med. Jour., 1935, i., 1126.
[4] Sulzberger, M. B., and Wise, F.: Arch. Derm. and Syph., 1931, xxiii., 519.
[5] Ingram, J. T.: Brit. Jour. Derm. and Syph., 1932, xliv., 422.
[6] Graham-Little, Sir E.: Brit. Med. Jour., July 20, 1935, p. 107.
[7] Some Pages from the History of the Prevention of Malaria. The Finlayson lecture for 1934 delivered before the Royal Faculty of Physicians and Surgeons, Glasgow. Glasg. Med. Jour., 1935, cxxiii., 49, 130, 202.

had wired ordering that the work in the port should come to a stop, a decision which Sir Malcolm was happily able to have put on one side. The proof of the value of his public health work there, judged by the touchstones of hospital and laboratory, was seen between 1901 and 1905 in a fall to one-eighth in the deaths from fever and to one-third in those from other diseases. This last effect was completely unlooked for and a special point is made of it, but the parallel outcome in the case of hookworm infection will come to mind. Sir Malcolm put to Wickliffe Rose of the Rockefeller Foundation the suggestion that too much was being made of the hookworm in the tropics—seeing that in his experience 50 worms, the number usually present at post-mortem in Malaya, did no damage to the man who harboured them—and that the pushing of a campaign against this infection at the same time as one against malaria would be too much for the Malayan mind; and he then notes a finding from the Report of the Uncinariasis Commission to the Orient [8] that fewer than 100 hookworms did no damage to the infected person. It is true that in a group from a penal institution this was so, but the report says clearly that in the kampongs there was "a progressive fall in hæmoglobin values from the 100-worm group to the 0-worm group." The other side of the question is seen in the American experience in Bilibid prison after Manila had been taken from Spain; its death-rate could not be reduced below 7·5 per cent. till they got rid of the hookworms and other intestinal parasites, which the prisoners harboured; after that there was a fall to 1·35 per cent. Moreover, from Clayton Lane's recent review in our columns [9] it is clear that even to-day there is no knowledge of the lowest weight of hookworms under which man is undamaged, though he believes it is certainly under 50. When malaria and hookworm are both present in any country, only those in authority on the spot have the knowledge necessary for the giving of a decision as to which should be attacked first. But the second danger is still there and must be kept in mind.

As its title tells, most of Sir Malcolm's lecture is a discussion, and a very useful one, of the history of the control of malaria in different parts of the world by biological methods, and by the production of delicate chemical changes in environment. It contains word-pictures of Gorgas and Darling, recounts the founding of the Ross Institute and its incorporation in the London School of Hygiene and Tropical Medicine, and comes to an end with a note on Ross himself, and a statement of the belief that in his last years he felt that the world was taking advantage of his discovery and that he had not toiled and fought in vain.

THE CHARAKA CLUB

IT is four or five years since a volume of the Transactions of that admirable literary society, the Charaka Club,[10] has appeared. They have always been a delightful medley of wit and wisdom, and those who are fortunate enough to possess the previous issues will be delighted to find that the literary vitality of the club is admirably preserved. The contents of the volume, as of its predecessors, are extremely varied, and of these the following may be mentioned as examples: Dr. S. W. Lambert supplies a learned reading from Vesalius, with facsimiles of the second printed edition and many illustrations. Dr. G. L. Walton has an essay on composition with the camera, admirable photographs explaining how an inside knowledge of photographic technique may assist the intensive study of pictures. Dr. Francis Packard introduces us to the earliest surgical case book of the Pennsylvania Hospital. The institution was among the first, or the first, that was founded as a hospital in the modern sense of the term, but it was not till some 50 years after its foundation that the management ordered that any records should be kept, and the original case reports run from 1803 for the next 25 years. The operations recorded are not those of the present day, mainly being amputations in the treatment of fractures and dislocations. Among the drawings at the end of the book are two representing the case of a double hare-lip before and after operation; the operation was performed by a Dr. Physik (his real name), and the results seem to have been good. Dr. Archibald Malloch writes on James de Berty Trudeau, artist, soldier, and physician, giving illustrations of his statuettes and bas-reliefs in bronze, some of them being piquant caricatures. Those who have the whole series of the Transactions will note with regret that they are reading the last contributions of Prof. George David Stuart, surgeon, wit, and accomplished poet. In a sympathetic chapter Mr. Bernard Sachs reminds us of his great personal and intellectual qualities, and his friends will endorse the tribute.

IS ENDOCARDITIS LENTA ALWAYS FATAL?

Endocarditis lenta (subacute bacterial endocarditis), though not a common disease, is relatively important, since it usually strikes young adults and, as many believe, is always fatal in the end. The fact remains that isolated cures are often reported, and one authority (Libman) even reports a recovery-rate of about 3 per cent. The possibility of applying effective treatment has lately been discussed by H. Schulten,[1] who points out that although endocarditis lenta is due to an infection of a heart valve by the *Streptococcus viridans*—or rarely some other organism—there are occasional cases, clinically very similar, in which blood culture is negative, and no bacteria can be found in the endocardial vegetations post mortem. Possibly the cause here is acute rheumatism, but whether or not this is so the recovery of such a case could not, Schulten thinks, be classed as a cure of endocarditis lenta; he seems to regard a positive blood culture as essential to diagnosis. Evidence of long-standing disease or congenital defect of the valves is also held as a rule to be a sine qua non, but it is believed that very exceptionally these may be absent—e.g., in endocarditis of the heart wall—or very slight. Schulten draws attention to another important feature of subacute bacterial endocarditis which may lead to a false idea of its curability—namely, the long remissions. It is now known that temporary improvement may be so great that the patient is able to resume his ordinary work, perhaps for several months; but almost without exception it is sooner or later followed by relapse and death. Treatment is divided by Schulten into two groups: (1) causal, which includes chemo- and sero-therapy, immuno-transfusion, vaccines, protein shock, and the surgical treatment of the presumed primary focus; and (2) symptomatic, including treatment for anæmia, the circulatory system, and antipyretic measures. In 23 of his own cases where a variety of these remedies were used, the patient always died. Replies to a questionnaire sent to his colleagues elicited a some-

[8] Rockefeller Foundation, Internat. Health Board. Publ. No. 9. 1920.

[9] THE LANCET, 1935, i., 1459.

[10] Proceedings of the Charaka Club. Vol. viii. London: Oxford University Press. 1935. Pp. 202. 25s.

[1] Med. Klinik, July 19th, 1935, p. 937.

what unusual result—namely, complete agreement on nearly all points. The general opinion was that an optimistic prognosis depended on a false diagnosis; that the mortality with or without treatment is 100 per cent., and that no therapeutic measure so far tried can alter the course of the disease. Two observers (Bingold and Lechleitner) make the suggestion that in the very early mild stage it may be amenable to treatment, but they offer no evidence in support of this belief. In short, the current opinion in Germany seems to be that the present measures used against endocarditis lenta are futile; and an important therapeutic problem remains wholly unsolved.

INJURIES OF THE CRUCIATE LIGAMENTS

IT is commonly assumed that the severest degrees of internal derangement of the knee-joint are those associated with rupture of the cruciate ligaments, partial or complete. These ligaments are so strong and their attachments so firm that there is reason to regard them as the main means of maintaining the stability of the joint. Superficial observation of the arrangement of the fibres would lead one to suppose that the anterior cruciate limits forward movement of the tibia on the femur, while the posterior limits backward displacement. Dr. Henry Milch,[1] of New York, however, thinks this a very imperfect view of the function of the cruciate ligaments. He holds that with the knee in full extension they have very little to do with stability, the collateral ligaments and the muscles being alone responsible. In flexion the action of the cruciates comes into play, some fibres of both anterior and posterior ligaments limiting backward and forward displacements on one another of the opposing surfaces of tibia and femur. Milch's observations on the results of rupture of the anterior ligament lead him to regard it as a structure of no very great importance. He has operated in cases of complete rupture of the ligament and has gone so far as to excise it completely, and he reports that its complete loss is compatible with relatively normal function of the knee-joint. Rupture of the posterior cruciate ligament is even less common, and he makes no definite pronouncement about its importance; but one gathers that he suspects this of being no greater.

The signs of rupture of the cruciate ligaments are said to be: (1) the possibility of passive displacement backwards of the tibia, or of its internal rotation, with the knee-joint in the position of extension; and (2) the possibility of displacing the tibia forward when the knee is flexed and the foot fixed. Of the first of these tests Milch says that it will never be positive if the collateral ligaments are intact; properly, therefore, it should be considered a test for injury of the collateral—more particularly the internal collateral—ligaments. The second test is reliable, he agrees, when present; but he found it positive in only 1 case out of 8. The probable explanation is that the tone of the quadriceps muscle is usually sufficient to mask the instability on which the test depends; under anæsthesia the muscle relaxes, and the "rocking" of the flexed knee-joint may then be demonstrated. The difficulty of distinguishing between injury of the cruciate ligaments and of the semilunar cartilages is shown by the number of times open operation has first revealed the lesion of the former. A tear of the internal meniscus may indeed be associated with rupture of the anterior cruciate ligament, and in one such case Milch excised both structures, with the result that on discharge from hospital the patient had a knee-joint with complete extension, flexion to 90°, and no abnormal mobility either anteroposterior or lateral.

In most cases of severe internal derangement of the knee-joint the disability, Milch believes, is due primarily to loss of integrity of the internal lateral ligament, and he recommends that treatment should be directed towards repair of this structure. In the worst cases this involves an open operation, with reconstruction of the ligament from the fibres of the fascia lata; but where the internal lateral ligament is intact he advises conservative treatment. One such case is cited in which even immobilisation was dispensed with. The diagnosis was rupture of the posterior part of the capsule, together with a tear of the anterior cruciate and fibular collateral ligaments, the last being shown by a possibility of hyperadduction to 15°. A knee cage brace was fitted, the sole and heel were raised on the outer border of the shoe, and the patient was allowed to walk about for six months. The result was good, and he now engages normally in sports.

Ski-ing accidents provide a number of these severe injuries to the knee-joint every winter, and it will be interesting to learn how far Milch's views on the relative unimportance of cruciate ligament injuries are upheld by other surgeons.

[1] Arch. of Surg., 1935, xxx., 805.

THE ESTIMATION OF IODINE

THE increasing attention which is being paid to the significance of small quantities of physiologically active materials is well exemplified by the case of iodine. The widespread occurrence of this element has long been known, but there has been a dearth of trustworthy data concerning its quantitative distribution owing to the lack of uniform methods of analysis. To remedy this state of affairs the Medical Research Council appointed a "Committee on Methods for the Estimation of Iodine," the expenses of which were defrayed by the Nitrate Corporation of Chile, Limited, and asked Mr. C. O. Harvey, a member of the Government Chemist's staff, to make the actual investigation. In his report[1] Mr. Harvey describes an improved and standardised method which he has devised, capable of giving consistent results when parts of the same specimen are tested by independent analysts. He sets out precise details for carrying out the analyses, with the various modifications required for the different materials analysed. Emphasis is laid on unsuspected pitfalls such as the necessity for employing pure nickel dishes in place of those containing copper alloyed with the nickel or even platinum dishes; strict precautions are necessary for avoiding contamination from impure chemicals or from laboratory air, and the titrations must be carried out on a bench not used for the other operations described. Promotion of further work on iodine in relation to the nutrition and health of man and animals, in which Mr. Harvey's method is likely to prove useful, has been entrusted to a new committee, appointed jointly by the Medical Research Council and the Agricultural Research Council. A reliable method of estimating the iodine content of the blood may well afford material for a more effective attack on hyperthyroidism.

THE death is announced on August 8th of Mr. Hugh Walker, ophthalmic surgeon to the Victoria Infirmary, Glasgow.

[1] M.R.C., Spec. Rep. Series No. 201. H.M. Stationery Office. Pp. 43. 1s.

PROGNOSIS

A Series of Signed Articles contributed by invitation

LXV.—PROGNOSIS IN CONGENITAL STENOSIS OF THE PYLORUS

In congenital stenosis of the pylorus we can recognise definite pathological changes, although the underlying cause is not yet clearly understood. The main feature, as is well known, is a marked hypertrophy or local gigantism of the circular muscle-fibres of the pyloric canal, which manifests itself by obstructive symptoms, usually at the age of about three weeks, and results in gradually increasing dilatation and hypertrophy of the walls of the stomach.

Of the two methods of treating congenital pyloric stenosis, recent experience has shown that medical treatment is uncertain in its results, requires a considerable period of time and, if unsuccessful, may lead to a grave state of malnutrition. It is now almost universally agreed that operation is the most satisfactory method of treatment, and we would emphasise, therefore, that in this article we are discussing the prognosis in cases treated surgically.

DIFFERENCE BETWEEN PRIVATE AND HOSPITAL CASES

The parents of an infant with pyloric stenosis demand first, an opinion on the chances of that particular infant surviving the operation; secondly, they are concerned with the ultimate prognosis, or, in other words, with the end-results of treatment, provided the infant survives the immediate risk. The factors governing operative prognosis in this condition are complex and probably not entirely understood, but it is the general experience that the outlook is much more hopeful in the private than in the hospital case. If we can find the reasons for this difference between hospital and private cases, we may be enabled to discover important factors which have a bearing on the immediate prognosis. How does the hospital case differ from the private case? At birth there is probably no difference at all, both infants apparently being strong and healthy, and not infrequently above the average birth weight. It is after the characteristic symptoms have commenced that the difference manifests itself. The mother of the infant which is ultimately brought to hospital is frequently harassed and preoccupied, and the fact that her baby is vomiting occasionally may not at first give rise to any serious concern. Eventually, perhaps on the prompting of a neighbour, she will decide that her breast milk is not agreeing with the child, and various feeds are tried in quick succession in a vain attempt to find something "which suits baby's stomach." The medical profession is not altogether blameless in this respect, since it is not always the interfering but well-meaning neighbour who advises the mother to wean her infant. Finally, after several weeks have elapsed, during which time the infant has been going rapidly downhill, he is brought to hospital in a state of extreme inanition, and operation is fraught with serious risks. It is this long delay which is so disastrous in a high percentage of hospital cases, since it means not only that at operation the child's resistance is at its lowest ebb, after weeks of starvation, but also that thereafter he is irrevocably deprived of his mother's milk which has long since dried up. It is little wonder that the mortality-rate in such cases is high.

The well-to-do mother has more leisure and can usually observe her infant more closely and, perhaps, though not always, more intelligently. Further, she is apt to be far more exacting in her demands on her medical attendant; he is likely to be summoned at the first sign of trouble, and she does not hesitate to demand a second opinion regarding her infant if she is not completely satisfied. This attitude reacts favourably on the infant, since it means that the pyloric obstruction will probably have been diagnosed and treated at an earlier stage than has that of the hospital infant, the child will not be so starved and dehydrated when it comes to operation, and, furthermore, the mother's milk will still be available at the critical post-operative period—a point of extreme importance. We believe that the mother is the best custodian of her infant in sickness or in health, and the very fact of removing an infant from its mother and placing it in the strange, somewhat impersonal atmosphere of a hospital, may do much more harm than is generally realised.

Here, then, are factors which may serve to guide us in giving an opinion regarding the immediate prognosis in any particular case. We must ascertain the duration and severity of symptoms, we must note the infant's general condition, and we must determine whether breast-feeding is going to be possible after operation. If these crucial questions have to be answered unfavourably, prognosis must be very guarded, since the mortality-rate may be 20 per cent. or even higher. If, on the other hand, the answers are favourable, we are justified in giving a favourable prognosis since, in our experience, the mortality-rate in this type of case is extremely low. In the experience of one of us (G. H.) over a number of years, the mortality-rate in hospital cases of pyloric stenosis is rather more than 20 per cent., whereas no private case has been lost, and we suggest that this remarkable difference may be explained to a large extent by the factors outlined above.

TECHNIQUE OF TREATMENT

The question of operative technique must, of course, have a bearing on the immediate prognosis, and although the Rammstedt operation, now generally adopted, is a comparatively simple procedure, it should, wherever possible, be performed by a surgeon who is experienced in children's surgery. Gentleness, combined with speed, is essential if post-operative shock is to be reduced to a minimum. A good deal of stress has been laid on the value of blood transfusion before and after operation to minimise shock and to increase the child's resistance, and in our experience this measure has been of distinct value in severe cases. Preliminary gastric lavage is desirable, and the parenteral administration of glucose-saline solution before and after operation is important owing to the dehydration which is invariably present to some degree. The great value of breast-feeding during the post-operative period has already been stressed, but if this is not possible, *frequent small* feeds should be given of some readily assimilable food such as peptonised milk. Nothing should be given by the mouth for four hours after operation. It is our practice then to give 2 drachms of glucose and water hourly for four to six hours, and thereafter an ounce of peptonised milk may be prescribed at two-hourly intervals; the amount of the feed and the length of the interval are gradually increased until by the third day after operation the infant is receiving a feeding appropriate to its age and weight. Close

attention to details of feeding and general management during the few days subsequent to operation is of extreme importance. The method of anæsthesia adopted does not appear to affect the issue, since in our series of cases both general and local anæsthesia have been tried and the mortality-rate has remained fairly constant. We do, however, favour local anæsthesia, supplemented, when necessary, by gas-and-oxygen.

POST-OPERATIVE COMPLICATIONS

Of the post-operative complications, the most frequent and most dreaded is *enteritis*. Here, again, it may be said that enteritis scarcely ever occurs in the private case, and its occurrence is probably governed by the same factors as influence operation mortality. Other complications such as *hyperpyrexia* and *hæmorrhage* are fortunately very rare, and their effect on prognosis is therefore *negligible*.

ULTIMATE PROGNOSIS

With regard to the ultimate prognosis in those infants who survive the operation and the post-operative convalescence, the problem is fortunately a simple one. We can confidently assure the parents that their child will grow up as any other child, and that no disability will mar its later life as a result of the congenital defect treated in infancy.

SUMMARY

At the present time the lives of many infants of the hospital class suffering from congenital pyloric stenosis are needlessly lost owing to long delay in diagnosis and unnecessary weaning, and until the possibility of this condition is considered in every infant with persistent vomiting, constipation, progressive loss of weight, and visible gastric peristalsis, prognosis must remain very guarded. The satisfactory results obtained with these cases in private practice, where circumstances permit of earlier diagnosis, serve to emphasise the fact that the mortality-rate for all cases is capable of being reduced to a very low figure, and we must hope that in the not too distant future it will be possible to predict with confidence a favourable outcome in the vast majority of infants with pyloric stenosis, irrespective of the class of society to which they belong.

GERTRUDE HERZFELD, M.B., F.R.C.S. Edin.,
Surgeon, Royal Edinburgh Hospital for Sick Children

H. L. WALLACE, M.B., F.R.C.P. Edin.,
Assistant Physician to the Hospital.

SPECIAL ARTICLES

THE EFFECT OF INFLUENZA EPIDEMICS ON THE CERTIFIED CAUSES OF DEATH

BY PERCY STOCKS, M.D. Camb., D.P.H.
MEDICAL STATISTICAL OFFICER, GENERAL REGISTER OFFICE, LONDON

THE idea that epidemic influenza usually occurs in a "setting" of associated strange fevers, agues, or dyspepsias finds a place in the writings of several epidemiologists and the association between influenza prevalence and that of certain other diseases with somewhat ill-defined respiratory, nervous, or gastro-intestinal symptoms has been stressed especially by Sir William Hamer.[1]

The 1918–19 epidemic in England and Wales was carefully studied in a Supplementary Report of the Registrar-General [2] with a view to leaving a permanent record of the manner in which deaths said to have resulted from influenza alone or in combination with other causes were certified, and a general broad estimate was also made of the contemporaneous deaths attributed to phthisis, pneumonia, bronchitis, and heart disease which probably resulted from the epidemic waves. This latter effect upon the certification of deaths from other causes individually could not be assessed from the experience of a single year, but experience has now accumulated of more recent epidemics—notably in 1922, 1927, 1929, and 1933, and it seemed worth while to try to determine with more definiteness what happens to the mortality as described on death certificates during the months when influenza is highly prevalent.

If influenza waves tend to be accompanied by an increased activity of other infections having some kind of affinity for influenza, or by a condition of the population rendering it more liable to succumb to other diseases, this should be evidenced by an increase over expectation in the mortality attributed to these other causes during influenza epidemics. The same thing would happen, however, with regard to chronic pathological conditions if superimposed influenzal infection frequently led to death which would otherwise have been postponed, provided that in many such cases the superimposed influenza was not recognised as such or not thought worthy of mention on the death certificate. If, on the other hand, there is any truth in the statement, sometimes made by persons who are themselves immune to influenza, that "an influenza epidemic can be created by talking about it," the implication being that once the medical profession has decided that an epidemic has begun many ill-defined causes of illness and death will be attributed to influenza, thus exaggerating the wave of prevalence or mortality assigned to it, we should expect to find that during epidemics the deaths attributed to such other causes would show a compensatory decline rather than an increase.

Collins,[3] in an instructive study of weekly influenza and associated mortality during 1918–29 in a group of 35 cities of the U.S.A., found that in the pandemic of 1918–19 about 8 per cent. of the excess mortality from all causes was credited to causes other than influenza or pneumonia, in the 1920 epidemic 23 per cent., and in the minor epidemics since 1920 about 40 per cent. About half of this excess mortality was attributed to other causes, with influenza or pneumonia stated as contributory, the remaining half having no mention of influenza or pneumonia, but being attributed to conditions such as heart disease, nephritis, cerebral hæmorrhage, phthisis, or bronchitis. As will be seen below, analysis of mortality in England and Wales during 1921–32 by months leads to a similar general conclusion, though the different precedence given in the two countries to influenza and pneumonia when stated in combination with other causes precludes exact

[1] Hamer, Sir W. H.: Proc. Roy. Soc. Med., 1931, xxiv., 1425; 1933, xxvii., 5.
[2] Supplement to 81st Annual Report of Registrar-General, 1920.
[3] Collins, S. D.: Public Health Reports, Washington, 1932, xlvii., 2159.

comparison. The present analysis, however, has been carried out for each of some 45 causes separately in the endeavour to ascertain which particular causes show the most pronounced sympathetic increase in mortality during influenza epidemics and which are unaffected, and to seek a reason for this.

Winter Influenza since 1920

Since the pandemic of 1918–19 influenza epidemics in England and Wales have shown a decided preference for the first quarter of the year, as may be readily seen from the diagrams for London produced by Mr. Spear in his recent paper.[4] In the 52 quarters from mid-1920 to mid-1933 the deaths assigned to influenza in England and Wales ranged from 2306 to 23,927 in the first quarters, but never exceeded 4015 in the second, 705 in the third, and 2362 in the fourth. Only in 1923 and 1926 were the first quarter's deaths exceeded by those in any other quarter (the second in each instance). Dealing with the separate months of the first quarter, the distribution of the 39 months of January, February, March, in the 13 years 1921–33 according to the number of influenza deaths which occurred is shown in Fig. 1 and Table I. To facilitate subsequent reference an arbitrary division has been made into "normal" months, defined as months having up to 2000 influenza deaths, "mild" epidemic months with 2001–4000, "moderate" epidemic months with 4001–7000, and "severe" epidemic months with over 7000 deaths.

TABLE I

Distribution of Months of First Quarters of 1921–33 *according to Number of Influenza Deaths (England and Wales)*

—	Deaths.	January.	February.	March.
"Normal"	Up to 1000	1921, 1923, 1926, 1930,	1923, 1926, 1928, 1930,	1923, 1930.
	1001–2000	1924, 1925, 1928, 1931.	1921	1921, 1922, 1926, 1928, 1933.
Mild epidemic.	2001–3000	1929	—	1932
	3001–4000	1927, 1932	1925, 1931, 1932	1925
Moderate epidemic.	4001–5000	—	1924	1927, 1931
	6001–7000	—	1922	1924
Severe epidemic.	7,001–8,000	—	1933	—
	9,001–10,000	1922	1927, 1929	—
	11,001–12,000	1933	—	1929

There were two Januaries in the moderate or severe categories, but February attained that level in five years and March in four. In 1933 January had 11,502 deaths attributed to influenza (the only month giving a higher total being March, 1929), whilst February recorded 7135 deaths.

When the 13 Januaries from 1921 to 1933 are arranged in order of influenza death-rate, it is found that the daily mean air temperature in England and Wales in the five months with highest influenza-rate was 39·4° F., whereas for the five months with lowest influenza-rate it was 42·5° F. For February the mean temperatures were, for the 5 highest influenza months 38·5° and for the 5 lowest 41·7°, whilst for March the corresponding figures were 41·9° and 43·6°. In view of this association between coldness and influenza mortality in the winter months, which might lead to erroneous conclusions, especially when respiratory diseases are under consideration, the analysis from which Fig. 2 has been drawn was carried out by separating the 36 months of the first quarters of 1921 to 1932 into two groups of (1) 18 warmest months for which the curves are continuous lines, and (2) 18 coldest months shown as broken lines. In Table II., however, each month, January, February, or March, has been treated separately and no division into cold and warm months has been made. These two analyses relate to the 12 years ending with 1932, the figures for 1933 not being available when the work was commenced.

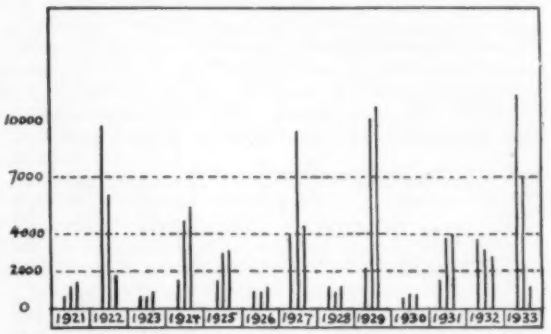


FIG. 1.—Influenza deaths in January, February, and March of each year 1921–33, in England and Wales.

METHOD OF ANALYSIS

The method of calculation was as follows. The number of deaths expected in each month was first calculated by assuming that the upward or downward trend of annual deaths from a given cause, owing to population increase, changes in prevalence, fatality, diagnostic accuracy or fashions in certification, proceeded in arithmetical progression during the period, and was represented by a straight line drawn through points having as ordinates the mean annual deaths in the quinquennium 1921–25 centred at 1923 and the mean annual deaths in 1926–30 centred at 1928. The total January deaths from the given cause in the decade 1921–30 were then assumed to be subject to the same fractional annual increment or decrement as the total deaths for the years as a whole—that is to say, the slope of the expected trend was assumed to be the same for each individual month as for the whole year. For a few causes, such as infantile diarrhœa, this would not be a safe assumption, but for the causes in Table II. there seems no reason why the general expected trend should be different for one month than for another. Thus the expected January deaths were represented by a straight line passing through a point whose ordinate, erected between 1925 and 1926 on the time scale, was equal to the mean January deaths in the decade 1921–30, the slope of the line being as determined above for the years as a whole. The annual decrements or increments thus calculated from the decade were assumed to continue constant until 1932, the straight line being prolonged to that year. Fluctuations above or below this expected datum line were assumed to be due to factors not progressively changing during the period but fluctuating from month to month and year to year, such as meteorological conditions and influenza epidemics.

At the head of Table II. the 12 years have been arranged in order of increasing influenza death-rate for the month in question and then divided into groups of three. Thus the order for the Januaries is 1930, 1923, 1921, 1926, 1928 and so on. The mean influenza-rate per million in each successive group of three months is then shown; for the Januaries it rises from 210 for the first group to 1735 per million for the last group. The actual deaths attributed to each cause in the Table were expressed as a percentage of the expected deaths in that month, and the figures shown in a given column are the means of the resultant percentage ratios for the three months named at the head of that column. That is to say, they are indices

[4] Spear, B. E.: THE LANCET, 1934, ii., 1331.

TABLE II

WINTER MORTALITY FROM VARIOUS CAUSES (PER CENT. OF THAT EXPECTED FROM THE SECULAR TREND), THE YEARS 1921 TO 1932 BEING ARRANGED IN ORDER OF INCREASING INFLUENZA DEATH-RATE FOR THE MONTH IN QUESTION

—	JANUARY.				FEBRUARY.				MARCH.			
Years forming group	1930 1923 1921	1926 1928 1931	1925 1924 1929	1932 1927 1922	1923 1930 1926	1928 1921 1925	1932 1931 1924	1922 1927 1929	1930 1923 1928	1926 1921 1922	1932 1925 1931	1927 1924 1929
Mean air temperature	43·3	40·5	39·5	41·3	41·5	41·8	38·8	38·0	42·9	43·3	40·5	42·9
Mean influenza death-rate	210	371	540	1735	247	555	1268	2819	296	466	955	2273
	Mean percentage ratio of actual to expected deaths.											
ALL CAUSES	86	99	99	116	83	88	107	131	89	93	102	122
All causes except influenza and respiratory diseases	95	97	99	110	93	100	109	110	95	104	99	105
*Whooping-cough	54	73	119	136	63	67	83	180	61	78	98	166
*Measles	89	159	94	79	134	94	120	81	180	67	112	93
*Diphtheria	113	91	84	98	97	103	84	109	97	120	78	84
*Acute poliomyelitis	83		102		90		100		106		83	
*Encephalitis lethargica	111		82		112		76		90		104	
*Cerebro-spinal fever	130		122		105		154		100		155	
*Cancer	98	101	102	99	98	100	102	101	101	99	99	100
*Respiratory tuberculosis	92	102	102	107	92	94	104	112	96	96	104	108
*Tuberculous meningitis	101	92	100	90	98	104	110	99	100	99	99	104
*Rheumatic fever	101	102	93	95	102	103	92	99	105	102	87	93
Erysipelas	102	110	89	94	100	107	84	101	95	98	116	101
Septicæmia, &c.	115	97	84	90	99	106	83	107	102	103	79	96
Rheumatoid and osteoarthritis	88	101	101	103	83	96	118	120	93	96	93	108
Diabetes	95	103	99	111	94	93	100	115	101	96	105	105
†Diseases of nervous system	95	101	96	102	94	97	100	110	96	96	97	106
Epilepsy	95	111	102	103	95	95	109	109	95	98	103	105
Diseases of ear and mastoid	105	106	96	96	103	96	101	100	99	107	94	101
Heart diseases	90	98	104	110	84	89	108	133	95	92	108	112
Aortic disease	89	107	104	102	96	94	101	107	98	97	105	104
Mitral disease	91	103	103	102	83	95	102	117	101	93	95	108
Fatty heart	87	99	107	100	90	90	106	116	96	92	97	112
Other myocardial disease	98	100	103	113	79	95	108	127	90	102	108	113
‡Cerebral hæmorrhage, &c.	92	103	112	104	92	94	104	111	96	94	108	108
Arterio-sclerosis	86	102	100	100	84	97	91	114	96	91	91	112
Respiratory diseases	72	103	107	126	69	75	111	149	80	81	103	145
Acute bronchitis	65	104	110	144	66	70	126	193	72	77	124	159
Chronic bronchitis	68	103	110	122	73	75	108	152	81	82	104	140
Broncho-pneumonia	69	102	104	131	64	72	113	158	73	80	95	156
Lobar pneumonia	84	101	106	107	83	85	101	125	95	83	97	120
Pneumonia not defined	80	99	106	122	75	79	104	152	80	80	91	124
Asthma	66	103	106	126	66	71	100	144	78	75	102	115
Gastritis	102	103	95	97	93	110	105	99	99	104	103	99
Gastric and duodenal ulcer	97	106	98	98	95	102	100	103	105	93	102	98
Appendicitis	98	108	105	98	94	101	105	105	105	95	96	101
Acute nephritis	93	104	105	103	104	98	94	103	97	97	96	104
Chronic nephritis	94	96	105	99	93	98	94	107	102	101	92	100
Cystitis, &c.	95	109	94	113	94	96	88	114	94	89	114	110
*Puerperal sepsis	101	117	99	89	100	105	99	99	105	106	105	93
Puerperal hæmorrhage	117	89	85	93	107	99	124	92	113	85	75	116
Puerperal albuminuria	104	76	94	100	90	106	86	99	101	113	81	89
Premature birth	99	102	98	112	92	99	112	108	96	100	109	107
"Old age"	84	100	104	114	85	88	105	127	88	92	108	120
*Suicide	104	98	96	106	105	100	100	98	101	109	89	100

* Usually taking precedence over influenza when associated with it as cause of death. † Excluding cerebral hæmorrhage.
‡ Including cerebral hæmorrhage secondary to arterio-sclerosis.

representing the mortality experienced (corrected for the general annual trend of population and other factors affecting certification of the particular disease during the period covered) in groups of months when these have been arranged in ascending series of their influenza death-rates. In similar fashion the graphs in Fig. 2 have been drawn by arranging the 18 warmest months, out of the 36 first quarters' months in the 12 years, in order of increasing influenza death-rate, dividing into convenient groups of three or more and plotting the mean of the percentage ratios of actual to expected deaths in each group of months against the mean monthly influenza death-rate for the group. The influenza-rate represents the number of deaths per 1000 living which would occur in a year if they continued at the same rate as during the month in question. The process was then repeated for the 18 coldest months, giving the second graph.

Influenza Certified as Joint Cause of Death

Before considering the results and what they signify it is necessary to understand what diseases take precedence over influenza when stated on death certificates jointly with it as the cause of death. Of the causes mentioned in Table II. (*a*), whooping-cough, measles, diphtheria, acute poliomyelitis, encephalitis lethargica, cerebro-spinal fever, puerperal sepsis, and suicide were generally given precedence over influenza when mentioned in conjunction with it, and also in the majority of cases tuberculosis, cancer, and rheumatic fever. On the other hand, deaths from (*b*) the other causes in Table II., if mentioned jointly with influenza, would in almost all cases be assigned to the influenza group. It follows from this that when influenza is epidemic additional deaths due to influenza being superimposed

on or complicated by any of the conditions (*a*), starred in Table II., would be assigned to the condition in question and would tend to enhance the deaths classed to it; and this would occur whether the associated influenza was mentioned on the certificate or not. Analysis of the deaths assigned to these causes during 1933 epidemic, in which influenza was mentioned as a complication, was carried out in order to throw more light upon the results for this group (see below). Additional deaths brought about or hastened by influenza complicating any disorders of the second group (*b*), not starred in Table II., would be either (1) assigned to influenza if that was mentioned as an associated cause, or (2) assigned to the other cause if influenza had not been recognised or mentioned as a complicating condition. In the event of alternative (1) the actual deaths classed to the cause in question would thereby tend to be *diminished* during influenza epidemics, but in the event of alternative (2) they would tend to be *increased* during epidemics. If a net increase is observed for one of these (*b*) causes it must be concluded that the number of additional deaths from that cause associated with the epidemic, but for which influenza was not recognised as a complication or not mentioned on the certificate as such, exceeded the number in which it was so recognised and mentioned.

Whooping-cough Mortality during Influenza Epidemics

Whooping-cough and measles present remarkable contrasts both in Table II. and Fig. 2, for whilst whooping-cough mortality was more enhanced by coincident influenza than any other of the causes examined, measles mortality was not enhanced, but on the contrary tended to be slightly less during influenza epidemics. The effect of influenza on whooping-cough deaths was evident alike in January, February, or March; thus in the groups of months of lowest influenza-rate the actual deaths were only 54 per cent. of those expected for January, 63 per cent. for February, and 61 per cent. for March, whereas in the groups of months of highest influenza-rate the corresponding percentage ratios were 136, 180, 166. This means that deaths attributed to whooping-cough were two and a half to three times as many during severe influenza epidemics as during the same months of the years when influenza was at a low level. Fig. 2 shows that this took place whether the months were warm or cold, and with a given influenza-rate it was not enhanced by cold but rather the reverse, as shown by the relative positions of the two graphs.

When the 36 months are grouped according to the number of influenza deaths occurring, the mean deaths per month attributed to whooping-cough are shown in Table III. to increase continuously from 350 in months having less than 1000 influenza deaths to 1536 in the month having 11,838 influenza deaths. In order to ascertain the rate of increase of deaths assigned to whooping-cough and some other causes, in Table IV. the columns of Table III. have been condensed into three groups, the first (A) consisting of the 19 months (shown in Table I.) with a "normal" influenza level of under 2001 deaths, the second (B) consisting of the 13 mild or moderate epidemic months with 2001–7000 deaths, and the third (C) of the 4 severe epidemic months with over 7000 deaths.

From Table IV. it appears that for every 100 deaths attributed to influenza in excess of the "normal," during mild or moderate epidemics 7 additional deaths, or during severe epidemics 9 additional deaths, were attributed to whooping-cough. These are average figures, the number of additional deaths in a given year being naturally dependent not only upon the severity of the influenza epidemic but also upon the number of whooping-cough cases present at the time. Thus at the end of 1932

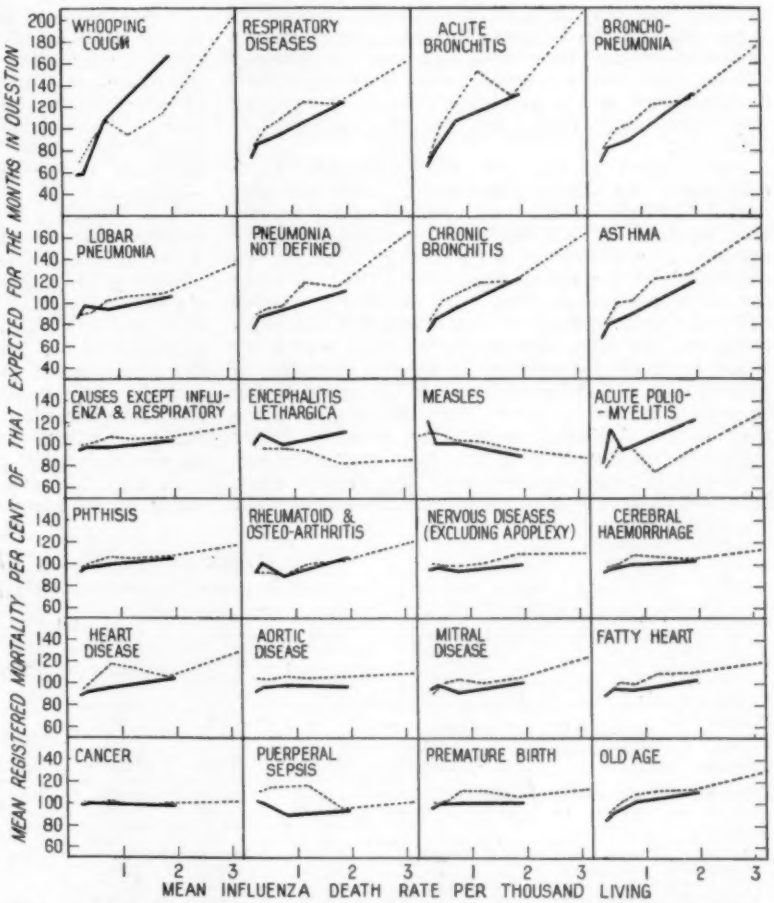


FIG. 2.—Effect of increasing influenza prevalence on the mortality attributed to various causes during the months of January, February, and March, 1921–32.

——— 18 warmest months (mean air temperature exceeding 41° F.)
..... 18 coldest months (mean air temperature 41° F. or below).
} Arranged in order of increasing influenza death-rate.

TABLE III

MEAN DEATHS PER MONTH WHEN THE 36 MONTHS OF THE FIRST QUARTERS OF 1921–32 ARE ARRANGED ACCORDING TO NUMBER OF INFLUENZA DEATHS

Influenza deaths	0–1000	1001–2000	2001–3000	3001–4000	4001–5000	6001–7000	9001–10,000	11,001–12,000
Months	10	9	2	6	3	2	3	1
Mean deaths per month.								
Influenza—								
with respiratory complications	490	937	1,688	2,401	3,170	4,408	7,362	9,236
without respiratory complications	307	483	677	1,011	1,259	1,794	2,354	2,602
Whooping-cough	350	450	491	505	577	943	1,122	1,536
Respiratory diseases (without mention of influenza)	7,445	9,150	9,670	9,560	10,795	13,798	16,778	17,405
Tuberculosis (pulmonary)	2,789	3,055	2,989	2,481	3,178	3,382	3,439	3,576
Diseases of circulatory system	8,317	8,678	13,615	10,756	10,285	8,275	11,548	15,012

whooping-cough was presumably at a low level when the influenza epidemic began, since only 421 deaths occurred in the fourth quarter compared with an average of 628 in the preceding 12 fourth quarters. The resulting excess in January and February, 1933, due to the superimposed influenza might therefore be expected to fall short of the 9 per 100 influenza deaths averaged during the severe epidemics of 1922, 1927, 1929. Actually, as shown in Table V., the excess of whooping-cough deaths resulting in the months of January and February, 1933, above the average for the same months of 1928 and 1930 (with influenza normal) was only 327, or 2 per cent., instead of 9 per cent. of the influenza deaths. For all causes combined other than influenza and respiratory disease, the observed excess was, however, almost exactly equal to the number expected by applying the percentage rates in the last column of Table IV.

Examination of the 801 whooping-cough death certificates for January–February, 1933, revealed only 22 with any mention of influenza as a complication, so it is evident that the excessive mortality attributed to whooping-cough during influenza epidemics is not for the most part recognised as being due to a double infection. (In the fourth quarter of 1918, with its 98,280 influenza deaths, 906 whooping-cough deaths were stated to be complicated by influenza in 119 instances.) It cannot therefore be proved that the influenza wave is the *cause* of the excessive mortality from whooping-cough, since they may both arise from some common factor unknown, but Fig. 2 shows that this factor could not be the rather greater coldness of the months when influenza happened to be epidemic. The explanation which most readily suggests itself is that the double infection is characterised by a very high fatality and is not usually recognised as anything other than whooping-cough with respiratory complications. The fact that in the 1933 epidemic both whooping-cough and respiratory disease mortality fell much below the expected figures, whilst other mortality reached the expected level, based on the previous severe epidemics of 1922, 1927, and 1929, suggests that it may be the *secondary* infections of influenza which spread to whooping-cough cases and prove specially fatal to them.

The absence of any similar consistent increase of deaths from measles or diphtheria during influenza epidemics suggests that the phenomenal increase of whooping-cough mortality is of real significance and of some epidemiological importance, and is not merely the outcome of unsatisfactory medical certification, which is liable to be blamed without adequate reason for anything which cannot be explained.

The occurrence of a wave of whooping-cough fatality contemporaneous with that of influenza is demonstrated in another way in Table VI., where the twelve years 1921–32 have been divided into the six years of highest influenza during the first quarter, indicated by +, and the six years of lowest influenza, indicated by −. The mean numbers of deaths during the first quarter, the two quarters coming before it, and the three quarters following, and the excess of the first quarter's deaths over the mean of those in the quarter immediately preceding and succeeding it are shown for each group.

In the "normal" years the average seasonal swing of influenza deaths was from about 600 in

TABLE IV

MEAN EXCESS OF DEATHS PER MONTH DURING INFLUENZA EPIDEMICS

	A. Normal months.	B. Mild or moderate epidemic.	C. Severe epidemic.	Excess of B. over A.	Excess of C. over A.	Excess over normal, per 100 influenza. During ordinary epidemics.	Excess over normal, per 100 influenza. During severe epidemics.
Months	19	13	4				
Mean deaths per month.							
Influenza, total	1,092	3,915	10,240	2,823	9,148	100	100
Influenza with respiratory complications — Number	702	2,777	7,831	2,075	7,129	73	78
Influenza with respiratory complications — Per cent.	64	71	76	73	78	73	78
Whooping-cough	397	587	1,226	190	829	7	9
Respiratory diseases (influenza not stated)	8,253	10,514	16,935	2,261	8,682	80	95
Tuberculosis (pulmonary)	2,915	3,043	3,473	128	558	5	6
Mitral disease	902	959	1,201	57	299	2	3
Cerebral hæmorrhage	3,035	3,407	3,660	372	625	13	7
"Old age"	2,322	2,503	3,141	181	819	6	9
Other diseases of heart and circulation	6,933	8,924	10,383	1,991	3,450	71	37
Other diseases	18,513	18,690	19,475	177	962	6	11
Total (not influenza)	—	—	—	—	—	190	177

the summer quarter to 3600 in the winter quarter, and of whooping-cough from about 700 to 1250. In the epidemic years, with a mean influenza peak going up to 15,000 in the winter quarter, the whooping-cough swing was increased to 653, 676, 2100, 1130, 559 for the five quarters from summer to summer. Measuring by this method the excess of the March quarter's deaths over the average of the December and June quarters, it is seen in the final column that an excess of 11,435 influenza deaths was accompanied by an excess of 850 whooping-cough deaths, or about 8 per 100, as found by the previous analysis (Table IV.)

Mortality from other Epidemic Diseases in Influenza Months

Table II. shows that the association of influenza epidemics with the mortality attributed to *diphtheria* was, if anything, negative during 1921–32. Thus taking the means of pairs of columns, January gives 102 for low influenza months and 91 for epidemic months, February gives 100 and 97 respectively, and March 108 and 81. Since diphtheria was given precedence over influenza when the causes were mentioned jointly, this cannot be due to peculiarities of certification. Out of 452 diphtheria deaths occurring during the 1933 epidemic of January and February, influenza was mentioned on four certificates only as a complicating condition, compared with 40 out of 1334 in the fourth quarter of 1918.

Measles manifested a similar negative association with influenza; thus taking the mean ratios in Table II. for the six low and six high influenza months, January gives 124 and 87, February 114 and 100, March 123 and 102. The inverse relation is shown also in Fig. 2, and is difficult to account for, since measles takes precedence over influenza. Only on three certificates was influenza mentioned in conjunction with measles in 1933. It may be that when influenza is very prevalent the physician called in to treat a child with broncho-pneumonia who is stated to have had measles, often prefers in the event of death to attribute the broncho-pneumonia to influenza than to the measles he did not see, but on this point I should like to have the opinions of practitioners.

Acute poliomyelitis deaths were somewhat more numerous when influenza was epidemic in January or February, but not in March, and Fig. 2 shows no evidence of increase save in the months of very severe influenza. There is no conclusive evidence of association owing to the small numbers of deaths and of months involved. Out of 202 deaths from acute poliomyelitis during 1933 influenza was mentioned on only one certificate.

Encephalitis lethargica deaths show no consistent relation with influenza, an apparent negative effect for January and February being offset by a positive one for March. Fig. 2 shows no effect whatsoever when the months are separated into cold and warm groups, but there is evident a curious excess of deaths in the warm over the cold months when months of equal influenza are compared, which accounts for the apparent negative relation with influenza when Januaries or Februaries of all degrees of coldness

TABLE V

Excess of Deaths during the Influenza Epidemic of January and February, 1933, over the Average Experience of 1928 and 1930 with Normal Influenza Incidence

—	Excess expected from number of influenza deaths.*	Actual excess over mean of 1928, 1930.	With mention of influenza on med. certificate.
Influenza	—	16,903	16,903
Respiratory disease (without mention of influenza) ..	16,058	7,410	—
Whooping-cough	1,521	327	22
Tuberculosis (pulmonary) ..	1,014	856	277
Mitral disease	507	260	—
Cerebral hæmorrhage	1,183	1,333	—
"Old age"	1,521	753	—
Other diseases	8,113	10,134	189†
Total (other than influenza and respiratory diseases) ..	13,859	13,663	488†

* By multiplying 16,903 by percentages in last column of Table IV. (based on 1922, 1927, 1929, experience).

† For diseases taking precedence over influenza only, including 99 cancer, 18 encephalitis lethargica, 17 syphilitic diseases, 6 deaths from violence, &c. (see text).

TABLE VI

MEAN NUMBER OF DEATHS IN THE FIRST QUARTER COMPARED WITH THOSE IN PRECEDING AND FOLLOWING QUARTERS, FOR (+) 6 INFLUENZA EPIDEMIC YEARS AND (−) 6 YEARS OF LOW INFLUENZA (1921–32). ALSO FOR 1933

	—		Third quarter preceding year.	Fourth quarter preceding year.	First quarter.	Second quarter.	Third quarter.	Fourth quarter.	Mean of quarters just before and after first.	Excess of first quarter over this.
1921–32.	Influenza.	+	573	1,977	15,163	2,429	533	1,771	2,203	12,960
		−	596	1,603	3,607	2,562	585	1,906	2,082	1,525
		Difference	−23	+374	+11,556	−133	−52	−135	+121	+11,435
	Whooping-cough.	+	653	676	2,100	1,130	559	514	903	1,197
		−	683	580	1,253	1,231	773	735	906	347
		Difference	−30	+96	+847	−101	−214	−221	−3	+850
	Tuberculosis (pulmonary).	+	6,567	7,522	9,442	7,951	6,447	7,237	7,736	1,706
		−	6,811	7,785	8,700	8,082	7,026	7,748	7,933	767
		Difference	−244	−263	+742	−131	−579	−511	−197	+939
	Respiratory diseases.	+	..	16,832	35,947	15,405	..	..	16,119	19,828
		−	..	18,198	24,223	15,873	..	..	17,036	7,187
		Difference	..	−1,366	+11,724	−468	..	..	−917	+12,614
	Mitral disease.	+	1,970	2,455	3,082	2,334	2,065	2,517	2,395	687
		−	2,000	2,508	2,610	2,226	1,918	2,457	2,367	243
		Difference	−30	−53	+472	+108	+147	+60	+28	+444
1933	Influenza		401	2,010	19,849	1,054	381	..	1,532	18,317
	Whooping-cough ..		574	421	1,071	466	380	..	443	628
	Tuberculosis (pulmonary)		5,793	6,448	8,619	6,746	5,533	..	6,597	2,022
	Respiratory diseases ..		..	12,752	26,458	9,754	..	..	11,253	15,205
	Mitral disease		2,076	2,576	3,147	2,188	1,870	..	2,382	765

are combined as in Table II. This disease and acute poliomyelitis are the only causes in Fig. 2 to show higher rates for the warm than the cold months. During the January-February influenza epidemic of 1933 influenza was mentioned as a complicating cause on 18 certificates out of a total of 179 deaths assigned to encephalitis lethargica, and in the remaining ten months of the year it was mentioned on six certificates out of 636.

Cerebro-spinal fever deaths were more numerous in the months of February and March when influenza was most prevalent, but this was not true for January. No conclusions can be drawn owing to the fact that a strong epidemic wave of the disease occurred during 1931 and 1932 after a period of comparative quiescence.

TUBERCULOSIS

Mortality from tuberculosis of the respiratory system increased steadily and consistently with rising influenza-rate, from 92 to 107 per cent. of expectation in January, 92 to 112 in February, and 96 to 108 in March (Table II.). The graphs in Fig. 2 bring out the slight but definite effect of coldness, but show the effect of influenza to be of greater importance than that of temperature.

Table III. gives the mean numbers of phthisis deaths in groups of months with increasing influenza, and Table IV. shows that during severe epidemics more than 500 deaths per month were registered from this cause in excess of expectation founded on normal periods. To every 100 additional influenza deaths during epidemics five or six additional phthisis deaths occurred. Table V. indicates that in January-February, 1933, with excess of 16,903 influenza deaths, the expected sympathetic excess of phthisis deaths over the "normal" level, 6 per cent. of 16,903 or 1014, was almost reached, the actual excess being 856 deaths. Examination of the total 6262 death certificates for tuberculosis of the respiratory system during these two epidemic months revealed a mention of influenza on 277, or about one in three of the additional deaths which were presumably brought about by the epidemic. (The excess of deaths attributed to phthisis calculated as due to influenza in the fourth quarter of 1918 was 3854, influenza being mentioned on 1619 of these.)

Table VI. shows that whilst the March quarters of the six low influenza years produced an average seasonal excess of 767 deaths over the mean of the preceding and following quarters, for the six epidemic years this excess was 1706 deaths. It also appears that whilst the September and December quarters following the epidemics had a deficiency of deaths averaging 1090 compared with the same quarters of the years with low influenza, the corresponding deficiency for the September and December quarters preceding the epidemics was only 507. This is no doubt due to the fact that influenza itself, or the secondary infections of the respiratory tract spread by those attacked by it, hastened the death of many phthisical patients who would otherwise have lived through another summer, thereby diminishing the deaths two or three quarters later to that extent. This compensatory effect is seen in phthisis mortality statistics for the period immediately following the pandemic of 1918–19 with its phenomenal phthisis mortality.

Tuberculous meningitis mortality shows in Table II. no evidence of any increase during influenza epidemics.

RESPIRATORY DISEASES (NOT TUBERCULOUS)

Deaths attributed to bronchitis, pneumonia, or other diseases of the respiratory system following upon or associated with influenza would be classified to a subgroup of influenza under the heading "influenza with respiratory complications," and would go to swell the total influenza deaths, but would not be included in the comprehensive group of respiratory diseases. If influenza was not mentioned they would be assigned to the respective respiratory diseases and the increase of these individual groups or of the total respiratory disease group during influenza epidemics represents deaths associated in some way with the influenza wave but not recognised, or at least not stated on medical certificates, as being caused or complicated by influenza.

Table II. shows that mortality from respiratory diseases as a whole (excluding those with mention of influenza, tuberculosis, and other primary causes such as measles) increased sympathetically with influenza from 72 to 126 per cent. of expectation in January, from 69 to 149 in February, and from 80 to 145 in March. The graphs in Fig. 2 indicate that this occurred at about the same rate in the warm as in the cold months, the effect of coldness being very small compared with the effect of influenza prevalence. Table III. shows that the mean monthly deaths from these causes progressively increased from 7445 in the ten months having less than 1000 influenza deaths to 17,405 in the worst epidemic month having over 11,000. In the same series of months deaths attributed to influenza plus respiratory disease rose from 490 to 9236.

From Table IV. it appears that whilst in the A group of normal months of the first quarter 64 per cent. of influenza deaths had respiratory complications mentioned, the proportion was 71 per cent. during mild or moderate epidemics and 76 per cent. during severe epidemics. (During the fourth quarter of 1918 the corresponding proportion was 86 per cent., and during the next quarter it was 83 per cent.) Of the deaths in excess of normal expectation during this last group of months, 78 per cent. had respiratory complications. For every 100 total influenza deaths in excess of normal there was, in addition to the 73 or 78 above mentioned (included in the 100) an excess of 80 deaths during ordinary epidemics or 95 during severe epidemics attributed to bronchitis, pneumonia, or other respiratory disease without mention of influenza. (The calculated proportions in the fourth quarter of 1918 and first quarter of 1919 were 20 and 38 per 100 respectively.)

Applying to the 1933 epidemic this expectation of 95 extra respiratory deaths to each 100 extra influenza deaths, Table V. shows the expected excess over the average for the corresponding months of the normal years 1928 and 1930 amounted to 16,058 respiratory deaths, but the total excess realised was only about half of this. By the alternative method in Table VI. in the six epidemic years the mean March quarter deaths amounted to 35,947 and the mean of the preceding and following quarters to 16,119, giving an excess, partly seasonal, of 1928, whereas for the other six years the corresponding excess, which we may regard as wholly seasonal, was only 7187. The difference of 12,641 accompanied a difference of 11,435 influenza deaths, or 111 to each 100. In 1933 the excess of March quarter deaths over the mean of the preceding and following quarters was 18,317 influenza deaths, or 16,792 more than in the low influenza years, leading to an expectation of an excess of $18,639 + 7187 = 25,826$ respiratory deaths over the preceding and following quarters, whereas the observed excess was 15,205. The percentage of respiratory complications amongst the influenza

deaths in this epidemic was, however, 77, the average proportion from preceding severe epidemic months being 76 (Table IV.).

Influenza is of course not the only factor concerned in producing monthly fluctuations in respiratory mortality, but it is undoubtedly the most important one under present conditions, the mean temperature having only a small effect in comparison, as Fig. 2. shows. Of the various respiratory conditions acute bronchitis shows the greatest sensitiveness to influenza prevalence, as also to coldness of the month in question (see Table II. and Fig. 2). Thus for January the mortality figure ranged from 65 to 144, for February from 66 to 193, and for March from 72 to 159 with rising influenza, a range similar to that of whooping-cough. Broncho-pneumonia is not quite so sensitive, and chronic bronchitis and asthma again slightly less. The close similarity of the "asthma" to the chronic bronchitis graphs in Fig. 2 is suggestive. All of these manifest a very high degree of association with influenza. Lobar pneumonia shows a much lower rate of increase, whilst pneumonia undefined gives graphs intermediate between lobar and broncho-pneumonia, approximating closer to the former than the latter.

A higher degree of association between the respiratory deaths and the influenza deaths is found to occur when the same months are associated than when influenza deaths in January are associated with respiratory deaths in February and so on, allowing a time lag of a month or more. Thus, if the Februaries are arranged in order of the influenza mortalities in the preceding month, the ratios of Table II. for respiratory diseases become 71, 86, 130, 117, instead of 69, 75, 111, 149, and for March arrangement in order of the preceding month's influenza produces the series 79, 87, 120, 123, instead of 80, 81, 103, 145.

TABLE VII

Results of Comparison with 1918–19 *expressed per* 100 *Influenza Deaths in Excess of the Normal*

—	1918 4th quarter	1919 1st quarter	1921–32. Severe epidemics.	1933. Jan.–Feb.
Influenza, excess of normal ..	100	100	100	100
Influenza without respiratory complications	14	17	22	22
Influenza with respiratory complications	86	83	78	78
Respiratory disease, so described, estimated as due to epidemic	20	38	95	44
Total respiratory disease due to epidemic	106	121	173	122
Ratio of this total to number of uncomplicated influenza deaths	7·6	7·1	7·9	5·5

Comparison with 1918–19 (Table VII.) indicates that the total increase in respiratory disease deaths during epidemics was seven to eight times the increase in uncomplicated influenza deaths, alike in 1918–19 and in the severe waves of 1922, 1927, and 1929, though in 1933 the ratio was lower. A great fall occurred, however, in the proportion of these extra respiratory deaths which were *stated* to be associated with influenza, from 83 per cent. (86 out of 106) in the fourth quarter of 1918 to an average of 45 per cent. in the later epidemics of 1922, 1927, 1929. In 1933, however, the proportion rose to 64 per cent., and it remains doubtful whether this fall represents a change in "fashion" of certification or a real change.

The accusation that the medical profession over-diagnoses influenza during epidemics receives no support from these figures, but it is unnecessary to conclude, either, that it is underdiagnosed, that is to say, that part or all of the extra deaths without mention of influenza did in fact result from a primary or complicating influenzal infection. If influenza is a virus disease and the respiratory complications result from secondary bacterial infections to which the respiratory tract is rendered more susceptible by the virus, presumably the secondary infection may be passed on to others after the virus has ceased to be transmissible, thus leading to contemporaneous waves of pneumococcal or other bacterial respiratory infections amongst persons who have escaped the influenza virus. This might also account for the rise in whooping-cough deaths, as suggested above.

HEART DISEASE AND "OLD AGE"

The graphs in Fig. 2 indicate a definite increase of deaths attributed to heart disease, without mention of influenza as a complicating cause during influenza epidemics, especially when these were severe and the weather cold. Table II. shows an increase from 90 to 110 for January, 84 to 133 for February, and 95 to 112 for March. This is evident, when no account of temperature is taken, for each heart condition in Table II., but separation of the cold from the warm months in Fig. 2 shows that for aortic disease the association with influenza is only apparent, being entirely explained by the consistently higher mortality in the colder months from this cause. The relation of sudden deaths to coldness of the winter months has been pointed out before.[5]

Mitral disease mortality is also affected by temperature, but shows an increase with influenza in the colder months. Table IV. indicates that to each 100 epidemic influenza deaths there were two deaths from mitral disease in excess of expectation during ordinary epidemics and three during the severe waves. Table VI. shows that in the 1933 wave there was an excess of 765 deaths from this cause over the average of the preceding and following quarters, compared with 687 in the six epidemic years between 1921 and 1932, and an average excess of 243 in six normal years, due to seasonal influences. On the basis of the influenzal deaths the anticipated excess of mitral disease deaths would be $243 + (18{,}317 - 1525)\ 444/11{,}435 = 895$, the actual figure being 765. There is no evidence from the Table that the increase during epidemics was followed by any such compensatory deficiency in succeeding quarters as was noticed for phthisis; mortality was enhanced during the half year after the epidemic rather than diminished.

The graphs for fatty heart closely resemble those for mitral disease. Deaths attributed to "myocardial disease" (other than acute myocarditis or fatty heart) have greatly increased in recent years, owing mainly to changes in fashion of certification of the deaths of old people. In 1923 they numbered 13,445 and in 1933 62,009. Satisfactory correction of mortality for this secular trend was therefore difficult, and the ratios in Table II. for this cause must be accepted with reserve, but they appear to show a similar degree of increase during epidemics as do the other heart disease groups. Table IV. shows that to each 100 "epidemic" influenza deaths there were not less than 71 additional deaths attributed to "other diseases of the heart or circulation" during mild or moderate epidemics, or 37 during the severe epidemics.

[5] Stocks, P.: Brit. Med. Jour., 1928, ii., 1188; Arteriosclerosis: A Survey of the Problem (E. V. Cowdry, Editor), New York, 1933, p. 212.

Deaths attributed frankly to "old age" without any circumlocution have been steadily declining, but after correction for this secular trend, the mortality figures in Table II. closely resemble those for myocardial degeneration, and the curves in Fig. 2 are similar to those for fatty heart, showing the double effect of coldness and influenza, of which the latter was the more important. According to Table IV., to each 100 epidemic influenza deaths, 6 additional deaths were attributed to old age during ordinary epidemics, or 9 during severe epidemics, and Table V. shows that during the 1933 wave there was an excess of 753 deaths attributed to this cause over expectation from the normal months of 1928 and 1930. The calculated excess was greater than this, but the downward trend since 1928 to the passing out of fashion of this form of statement has not been allowed for in Table V.

OTHER CAUSES

Combining all causes other than influenza and respiratory diseases, mortality was affected to the extent of a rise from 95 per cent. of expectation during the Januaries of lowest influenza to 110 in the Januaries of highest influenza. For February the corresponding indices were 93 to 110, and for March 95 to 105. Fig. 2 indicates that, whereas at a given influenza level coldness was a factor which increased this mortality to some extent, influenza was a more important factor, especially during the colder months. Table V. shows that the anticipated excess of 13,859 deaths from this total group of causes during 1933 epidemic over the same "normal" months in 1928 and 1930 was almost exactly realised.

Diseases of the nervous system, excluding hæmorrhage into the brain, showed a slight rise with influenza, at any rate in the colder months (Fig. 2). Cerebral hæmorrhage, including the deaths attributed to arterio-sclerosis as primary cause, increased more definitely, and it appears from Table IV. that to each 100 epidemic influenza deaths from 7 to 13 additional deaths from cerebral hæmorrhage occurred, the proportion being smaller during the more severe epidemics in contrast to most of the other causes in the Table. The excess of deaths in the 1933 wave, 1333, agreed fairly well with the calculated excess (Table V.). Arterio-sclerosis without cerebral lesion also increased with influenza (Table II.).

Cancer, a disease which was given precedence over influenza when jointly stated with it, manifests no association whatever with either air temperature or influenza prevalence. During January and February, 1933, 10,697 deaths from cancer occurred, and for only 99 of these was influenza mentioned as a complicating cause. (In the fourth quarter of 1918 the proportion was 53 out of 10,633.)

Mortality from nephritis failed to rise consistently with influenza, although January and March deaths from acute nephritis and January and February deaths from the chronic form manifested a slight apparent association with it.

Puerperal sepsis and septicæmia also show, if anything, a negative association with influenza prevalence, and for other puerperal causes there is no consistent change. Premature birth mortality tends to be slightly enhanced during epidemics, as might be expected.

Digestive causes show no association, nor does suicide.

According to Table IV., to each 100 influenza deaths in excess of the normal level, about 180 or 190 additional deaths from all other causes are certified during influenza epidemics, respiratory conditions generally forming a somewhat larger proportion of these during the more severe epidemics, though in 1933 this was not true.

Examination of all the death certificates during January and February, 1933, for the causes usually taking precedence over influenza when jointly mentioned with it (those starred in Table V., syphilitic diseases, scarlet fever, intestinal obstruction, and a few other causes, but excluding suicide or accident) revealed a total of 482 deaths with mention of influenza, or about 1 in 48 of the total deaths from these causes. The total of 482 consisted of 22 whooping-cough, 277 phthisis, 99 cancer, 18 encephalitis lethargica, 17 syphilitic diseases, 10 non-pulmonary tuberculosis, 7 premature birth, 8 intestinal obstruction, 5 puerperal sepsis, and 19 others. It must be remembered that an ordinary attack of influenza more than two or three weeks prior to death from one of these causes would not usually be mentioned on the death certificate, so the above finding could only be taken to mean that about 1 in 48 persons living were attacked on the average in each two or three weeks during the two months, or about 1 in every 12 to 18 during the whole epidemic. The Scottish figures [6] for the insured population show that during the year ending June, 1933, 6 cases of influenza occurred to each 100 persons, which suggests that between 1 in 10 and 1 in 20 persons were attacked during the epidemic. The incidence rate recorded for the army in the United Kingdom in 1933, was 1 in 25. The prevalence suggested by death certification is therefore in reasonable agreement with such measures of the actual incidence as are available.

Summary and Conclusion

During the influenza epidemics of 1921–33, which occurred mainly in the first quarter of the year, monthly mortality certified as due to many other causes, without mention of influenza as a primary or contributory cause of death, increased step by step with the severity of the influenza mortality. The causes most remarkably affected were whooping-cough, acute bronchitis, broncho-pneumonia, chronic bronchitis, asthma, pneumonia not defined, lobar pneumonia, and pulmonary tuberculosis, but mitral disease, fatty heart, myocardial degeneration, old age, cerebral hæmorrhage, arterio-sclerosis, nervous diseases, and premature birth also showed pronounced increase of deaths during epidemics.

During moderate epidemics each 100 influenza deaths, so described, in excess of the number occurring during the same months in interepidemic periods, included 73 with respiratory complications, and was accompanied by 190 other deaths in excess of expectation, consisting of 80 from respiratory diseases (without mention of influenza), 13 from cerebral hæmorrhage, 7 from whooping-cough, 6 from "old age," 5 from pulmonary tuberculosis, 2 from mitral disease, 71 from other diseases of the heart and circulation, and 6 from other conditions. During severe epidemics each 100 influenza deaths was usually accompanied by rather more than the above proportion of deaths from the respiratory causes, offset by less from circulatory causes.

Whooping-cough showed the most phenomenal rise of mortality with influenza in proportion to its numerical importance as a cause of death, and it is suggested that either the double infection must be very fatal, and must generally pass unrecognised as such, or else the increased mortality must be due to

[6] Report on Incapacitating Sickness in the Insured Population of Scotland, 1932–33, Table V.

the secondary respiratory infections being transmitted from influenza to whooping-cough patients.

It is also suggested that the great increase in deaths from bronchitis and pneumonia (without mention of influenza) during epidemics need not indicate lack of precision in diagnosis, but may indicate contemporary waves of the secondary infections of the respiratory tract started by persons originally infected by the influenza virus. The heavy phthisis mortality was partly attributable to hastening of inevitable death from that cause, and to that extent was offset by diminished deaths in the latter half of the year.

There was no clear evidence of increase of infections of other parts of the body, and the large increase in deaths attributed to non-respiratory causes during influenza waves seems to require either (1) that there is a lowered state of resistance to many disorders at the time when the epidemics occur, or (2) that influenza as a cause of death is very often not diagnosed owing to the medical attendant not having been consulted in the early stages of illness but only when sequelæ have developed, or (3) that influenza though recognised as the initial cause is not mentioned as such on the death certificate.

After giving full weight to the possible alternative explanations, there can be no doubt that incomplete medical certification is partly responsible for the widespread diffusion of the increase in mortality during influenza epidemics over the list of causes, and particularly for the great increase in deaths ascribed to myocardial disease and old age. It was hoped that the introduction of a new form of certificate in 1927 would result after a short interval in a very definite improvement in the reliability of medical certification, but whilst this hope has been fulfilled in certain respects it has not yet been realised to the desired extent in other directions, especially with regard to the manner of statement of joint causes of death.

The writing of a satisfactory death record is often a matter of no small difficulty, and it is to be regretted that the representations made some six years ago that adequate instruction should be given to every medical student in the matter of death certification have not yet met with a wider response in the teaching schools. A considerable service can be rendered to the preventive medicine of the present and of the future by each physician, surgeon, or pathologist who endeavours to produce accurate and complete mortality and notification records, and time can surely be found even in an overloaded curriculum to impress this fact upon every student before training is completed.

MEDICINE AND THE LAW

Omission to Notify Infectious Disease

A DOCTOR was fined sums amounting (with costs) to £6 5*s*. at Daventry last month for failure to notify cases of infectious disease. The alleged offences related to dates in March, April, and June, and the solicitor who prosecuted on behalf of the rural district council stated that on Jan. 10th the doctor's attention was called to the necessity of notification. The medical officer of health gave evidence that no certificates had been received in respect of three scarlet fever patients, a certificate for a girl suffering from diphtheria was not received until after she had recovered and left hospital, and in another scarlet fever case, admitted to hospital on June 1st, notification was not received till June 18th. The doctor pleaded guilty. In extenuation it was said that he had been overworked at the time and that three of the cases came from households where previous cases of infectious disease had already been notified. It was suggested that in some districts it is the practice to regard the notification of a single case from a particular family or home as covering subsequent cases from the same family or home. The Public Health Acts give no support to such a practice.

Alleged Attempt to Commit Suicide by Starvation

At Blackpool police-court last week Mr. Davidson, the former rector of Stiffkey, was charged with unlawfully fasting "with intent feloniously, wilfully and of his malice aforethought, to kill and murder himself." Apparently the accused was exhibited in a cubicle at a wax-works exhibition with posters which described him as intending to starve himself to death if an adverse decision of the ecclesiastical court were not reversed and if he were not reinstated by the bishop. A police officer said that the accused was so weak after a fast of ten days that he had to be lifted out of the case. It was contended on behalf of the defendant that there was no evidence that in this ten-day period he had been in any danger of death. The magistrates thought there was a case to answer and committed him for trial at Preston quarter sessions, bail being allowed on the defendant undertaking that meanwhile he would not starve, pretend to starve, or exhibit himself in a cabinet.

The law against attempted suicide is variously enforced. There have been official announcements that in many cases the police take no action unless it is necessary in the interests of the accused. The "hunger-strike," undertaken for political motives or otherwise, has no immunity from prosecution. The motive of attempted suicide is immaterial.

IRELAND

(FROM OUR OWN CORRESPONDENT)

SWEEPSTAKE MONEYS

IN answer to a question addressed to him in the Dáil the Minister for Local Government and Public Health has given some interesting information about the money which has been distributed to the hospitals of the Irish Free State from sweepstakes funds and the money which awaits distribution. He states that there is at present in the hands of the National Hospital Trustees a sum of £4,050,778, to which is to be added approximately £354,835 derived from the sweepstake on the Derby of this year, not yet paid over. Beside these sums the Minister has in his own hands for distribution £433,177 obtained from sweepstakes held before the passing of the Hospitals Act, 1933. So far sums amounting to £3,703,080 have been distributed to the hospitals under various Acts. He added that a preliminary report has been submitted by the Hospitals Commission and is under consideration. There has been, and will be, no avoidable delay in carrying out the intentions of the Public Hospitals Act, 1933, but obviously the matter is one of such magnitude that its investigation will take time.

THE LATE DR. C. M. O'BRIEN

Dr. Christopher Michael O'Brien, who died in Dublin on August 5th, qualified from the Royal Colleges of Ireland in 1892 and 1893, and graduated as M.D. Durh. in 1900. Early in his professional

career he became interested in dermatology, and was the first medical man in Ireland to practise that branch of medicine as an exclusive specialty. He founded the Skin and Cancer Hospital in Dublin while he was still a young man, and continued to conduct it up to the time of his final illness. He was one of the first in these countries to apply radium to the treatment of disease. A kindly and generous man he was held in much esteem by his friends and patients.

VIENNA

(FROM OUR OWN CORRESPONDENT)

REFORM OF THE MEDICAL CURRICULUM

THE reforms which the present authoritarian government is introducing into every branch of public life have now been extended to the universities, where the following new regulations will come into force next session. Each academic year is to consist of two semesters (or terms), the winter semester lasting from Oct. 1st to Jan. 31st and the summer semester from Feb. 15th to June 28th. Thus both terms will be of about the same length, whereas under the present system the winter term has been five months and the summer term only three months. The dates given mark the commencement of lectures for students. The Christmas vacation will be from Dec. 19th to Jan. 7th and the Easter one from March 16th to April 20th. Examinations and graduations (called here "promotions") will be held if necessary all the year round, except during the Christmas vacation and the summer vacation (July 15th to Sept. 20th) when all the universities will be closed. During these "free weeks" the students will be expected to take part in "university camping," which is a mild form of military training. Regulations for these exercises will be published shortly. Various special regulations are also made for the maintenance of hospital and ambulance services and of scientific work during the vacations.

Changes have also been made in the existing rules governing medical examinations, which have been in force since 1903. Medical students must henceforward pass three groups of examinations, each of which is termed a ***Rigorosum***. The first rigorosum is passed in the fifth semester, and consists of natural science (physics), chemistry, physiology, histology, and anatomy. There will be theoretical (viva voce) and practical examinations in all the subjects except natural science, in which the examination will be purely theoretical. Biology has been dropped, and students who have already taken the first rigorosum but have failed in biology will not be required to sit again in that subject. The second rigorosum will consist of examinations in morbid anatomy and histology, pharmacology and dispensing, materia medica, pædiatrics, neurology, and psychology. Candidates will be examined theoretically and practically in all subjects except pharmacology and dispensing, for which only a theoretical examination is required. General and experimental pathology, which was formerly also included in the second rigorosum, has been dropped. The third rigorosum, taken after the eleventh semester, embraces all the other subjects of the medical curriculum (surgery, including orthopædics; obstetrics and gynæcology; ophthalmology; oto-rhino-laryngology; dermato-syphilology; hygiene and forensic medicine).

A novel feature in the new regulations is the clauses directed against unsuccessful candidates. Under the old regulations a candidate who failed in part of the first rigorosum was allowed to take the examination again several times if necessary. Now he is only allowed three attempts if he fails in one subject, and two if he fails in two or three subjects. Should he fail in four subjects he will not be allowed to continue the study of medicine in this country, nor may he later apply for registration in Austria on the strength of a foreign medical qualification. A candidate who has been unable to finish his first rigorosum within two years of first presenting himself for the examination will also be permanently excluded from medical study in Austria. Exceptions will naturally be made where the candidate is not responsible for the delay (illness, war). A candidate who has successfully passed all three examinations must apply within six weeks for "promotion" to the degree of "doctor universæ medicinæ." Otherwise he runs the risk of losing his turn and may have to wait for months before he is called up.

The idea of eliminating the less successful students during the course of their medical studies is by no means unreasonable. Its aim, of course, is to prevent students from embarking on a career for which they are not suited. The necessity of finishing their studies within a limited time will encourage undergraduates to concentrate on their work, and the majority of the profession approve of the new rule. The *sine qua non* is that the examiners deal fairly with the candidates, and this is certainly to be expected in Austria.

BIRTH-RATE AND INFANT MORTALITY

Changes in the population have shown the same trend during recent years in all the countries involved in the late war. After 1918 the birth-rate went up, only to fall again a few years later. In Austria in 1920 there were 137,372 live births, in 1921 there were 141,834 (an increase of over 3 per cent.), and in 1922 there were 150,958 (an increase of 7 per cent.). But that was the peak year, and the following Table for the next ten years shows a decrease of 40 per cent.:

Year.	Live births.	Year.	Live births.
1923	146,633	1929	110,171
1925	135,594	1932	102,254
1927	118,302	1934	90,934

Some countries have attempted organised measures to increase their birth-rates (premiums to families with numerous children), but here we have only one scheme of this kind: a premium of 120 Austrian shillings (£4 10*s.*) is paid for a live birth to mothers insured under the new Social Insurance Act. Attempts to bring down the infant mortality have however given satisfactory results. In 1920 it stood at 157 deaths per 1000 live births; next year it dropped to 154; and since then it has constantly decreased, as is seen from the following Table of mortality among infants up to twelve months old:

Year.	Total deaths.	Deaths per 1000 live births.	Year.	Total deaths.	Deaths per 1000 live births.
1923 ..	20,653	141	1930 ..	11,665	106
1925 ..	15,895	117	1932 ..	10,888	106
1928 ..	13,964	120	1934 ..	8,821	91

The rapid drop in the total number of deaths is of course due to the decrease in the birth-rate, but the drop from 141 to 91 deaths per 1000 births is undoubtedly satisfactory. The decrease is due to

the work done by the executive committee for infant welfare instituted by the ministry for social welfare and public health. They began with 104 clinics which gave advice to mothers. A special scientific committee composed of the leading pædiatricians, directors of the local boards of health, and officers of the ministry investigated the problems of protection of mothers and infants against disease, and laid down general principles for the guidance of the clinics. It also assisted the local authorities in organising similar clinics all over the country, so that now we have 492 advisory centres with 78 branches and 13 antenatal clinics scattered over Austria. A staff of 515 doctors and 530 trained nurses is employed, and the expenditure for 1934 was 773,000 shillings (£30,000) and an attendance of 47,286 infants under eight months and 56,112 over eight months is recorded.

PANEL AND CONTRACT PRACTICE

The New Insurance Act Explained

JUST before Parliament rose Royal Assent was given to the National Health Insurance and Contributory Pensions Act, 1935, and on the same evening the Minister of Health broadcast an explanation of the chief features of the Act. It may be well briefly to recapitulate the principal provisions which affect the medical profession.

On ceasing to be insurably employed all insured persons are to have a free insurance period on an average of 21 months. During this period insured persons are to be protected for all pensions purposes and entitled to all health insurance benefits—medical, sickness and disablement, maternity, and additional benefits. If at the end of the free period an insured person who has been continuously insured for ten years, up to the date when he ceased work, proves that throughout the period he (or she) has been unable to obtain insurable work his insurance will be extended so long as unemployment continues to be proved year by year. During these periods of extended insurance he will be covered for all pensions rights and be entitled to medical, maternity, and additional treatment benefits, and will remain a member of his approved society. He will not be entitled to sickness and disablement benefit, but he can regain title to these benefits at the full rates if he again obtains insurable employment, although of course not immediately.

Under the Act of 1932 many insured persons have already ceased to be entitled to medical benefit and but for the passing of the new Act would have lost their pension rights at the end of 1935. These persons will be subjected to the ten years' test, but for the great majority of them the test will be applied at a much later date than that on which they ceased to be employed. If they satisfy the test they will be maintained in insurance so long as they continue to prove unemployment and, as from Jan. 1st, 1936, will be restored to medical, maternity, and additional treatment benefits and their pension rights will continue. Insurance doctors, especially those in the depressed areas, will thus have restored to their lists many of the insured persons removed under the 1932 axe. As from July, 1934, all arrears due to proved unemployment are to be excused in full; half the cost will be met by a levy on each contribution paid by members of approved societies and the other half by an annual Exchequer grant.

Hitherto deposit contributors have ceased to be entitled to medical benefit when their individual credit in the deposit contributors' fund becomes exhausted. In future, arrangements will be made to provide medical benefit for life to deposit contributors who are entitled to the benefit on reaching the age of 65, and they will thus be treated, for medical benefit, similarly to members of approved societies attaining that age.

Another anomaly which will be removed is in respect of a woman aged over 65 who marries. In the past such a woman, who otherwise would have been entitled to medical benefit for life, upon marriage was treated as having become a "Class K" member, and lost her title to medical benefit in about eighteen months.

Assistance at a Vaginal Operation

A west-country insurance practitioner assisted at an operation on one of his patients in which the cervix uteri was amputated and the vagina narrowed by stitches fore and aft. The insurance and local medical committees were in agreement that this assistance was of a specialist nature and that the doctor had the necessary skill and experience for its performance, but the Minister thought that it came within the doctor's terms of service and ordered an inquiry. On behalf of the two committees it was urged that the assistant at such an operation might be called upon to arrest a sudden hæmorrhage, or to hold the artery forceps or retractors in such a way as to enable the surgeon to get a clear view of the field of operation, and that many surgeons would prefer the ward sister as an assistant because of her greater familiarity with the task. It was further suggested that after years of absence from operating theatres some practitioners would not possess experience of modern operative procedure. A questionnaire had been sent to the 154 practitioners on the insurance committee's list to which 106 had replied, 48 of them being surgeons on the staff of their local hospital. Eight of these surgeons said they would be prepared to accept as an assistant at a major operation every insurance practitioner in the area from which his surgical cases were derived; the other 40 would not. Of the 58 other doctors who replied 25 would be prepared to act as assistant at a major operation—8 of them habitually act as such at all major operations performed on their insured patients; 33 would not be prepared to act; and 50 do not act as assistant at major operations.

The referees report that they are satisfied that the treatment in question did not involve the application of special skill and experience of a degree or kind which general practitioners as a class cannot reasonably be expected to possess. They add that the fact that 40 of the 48 surgeons on the staff of their local hospital who replied to the questionnaire were not prepared to accept as an assistant *every* panel practitioner in their area is in their view irrelevant to the inquiry. The issue was whether general practitioners as a class can reasonably be expected to possess the necessary skill and experience, not whether every general practitioner possesses it or is thought by his fellow practitioners to possess it. The fact that 33 of the 154 doctors to whom the questionnaire was sent said they would not be prepared to act as assistant at a major operation was too vague without knowing what operation

they had in mind. The reason why so few habitually did act as assistants at all major operations appeared to be because the hospital where the operation occurred was frequently outside their area.

While it must be remembered that the decision relates only to the particular case the observations can well be applied as a general principle.

A Remitted Penalty

A month or two ago (see THE LANCET, May 11th, p. 1129) the London local medical and panel committee investigated a complaint that a doctor had secured the transfer to his list of a patient of another doctor without the knowledge or consent of the insured person. The complaint having been found to be established it was necessary, under existing rules, for the committee to inform the Minister that the continuance of the doctor on the Medical List would be prejudicial to the service. Subsequently, however, it was felt that in the event of the Minister confirming the finding of the committee the penalty of removal from the list would be excessive. Only one such misdemeanour was alleged, and the complainant wanted to withdraw his representation provided the respondent sent a suitably worded undertaking not to offend similarly again. The respondent having now written expressing regret, and undertaking that there shall be no recurrence of the offence, the committee have decided (subject to a verbal amendment of the letter) to comply with the complainant's request.

It may be recalled that, after an investigation by its medical service subcommittee into any question relating to the conduct of a practitioner, an insurance committee may take action in any one or more of the following ways:—

(1) If they are satisfied that owing to the number of persons included in his list the practitioner is unable to give adequate treatment to all those persons, they may, after consultation with the panel committee, impose a special limit on the number of persons he may have included in his list.

(2) They may recover from him any expenses (other than those incurred in connexion with the medical service subcommittee's investigation) which have been reasonably and necessarily incurred by the committee or by an insured person or his representative owing to the practitioner's failure or neglect to comply with the terms of service.

(3) They may recommend the withholding of money.

(4) They may make representations for the removal of the practitioner's name from the medical list.

But it not infrequently happens that a practitioner is found to have committed an error of omission or of commission which is hardly of such a nature as to merit action under any of these four heads. It has become the custom for insurance committees in such cases either to warn the doctor of the necessity for a strict compliance with his terms of service, to caution him or to censure him. But it must be admitted that a censure is usually associated with action under (2) or (3) or both.

A Suspended Report and its Sequel

Committees are generally believed to have less compassion than their individual members. The recent action of a provincial medical benefit subcommittee reveals a very human touch; the pity of it is that the endeavours of the committee to help a doctor did not meet with success. In February last the committee investigated two complaints of failure to visit two persons on his list when requested to do so. The doctor admitted the failure which he ascribed to illness; he was, he said, undergoing a course of special treatment which would last five months. The committee decided to defer submitting their report on these two cases for six months conditionally upon the doctor undertaking to furnish at monthly intervals certificates (1) from the specialist providing the treatment that the doctor was following his directions (2) from a colleague that he was diligently carrying out his duties as an insurance practitioner. Article 33 (4) of the Regulations requires a medical service subcommittee to draw up a report and to present it to the insurance committee, but it does not specify when the report is to be presented, and there was nothing irregular in keeping the report in suspense to see how things turned out. Unfortunately the doctor failed to furnish the certificates required and it became necessary for the subcommittee to report that he had absented himself from his practice for longer than a week without first informing the committee as required by Clause 11 (2) of the Terms of Service, and to make a recommendation as to the action to be taken. The doctor then asked for leave to withdraw his name from the Medical List, and this was granted subject to an undertaking (which was given) that he would not, without the Minister's consent, engage in insurance practice as a principal.

PUBLIC HEALTH

Gas Heating of Flueless Rooms

THE tendency for houses to be built with some of the rooms unprovided with chimneys no doubt makes for economy in building—a matter of importance in these days when the costs of building are so high. The advisability, from a health point of view, of this practice is however a serious question which is so far from having been settled that the governor of the largest gas company in the world has induced the Royal College of Physicians to refer it to a committee of physiologists and public health experts. The conclusions so far reached by this committee have been made public, pending a final report. They refer only to conditions in dwelling-houses and not to those in factories and workshops.

At the outset the committee are faced with the fact that the rate of exchange of air in a room ventilated only by imperfectly fitting doors and windows, with or without the occasional air brick, is not really known with any precision; it is surmised that the air of such a room may be renewed from once in two hours to once in an hour, but whether this estimate is approximately correct, or whether even the higher rate presumed is sufficient to keep the room in a healthy and comfortable condition is not known. It is hoped by the committee that experimental work may be arranged on this point. With unventilated rooms the escape of heated air and the entry of cool air, which in cold weather needs to be heated, will be relatively small and therefore the amount of heating will be proportionately less than in rooms with chimneys or open windows; but the use in any room of flueless gas heaters, which discharge all the sensible heat and also all the products of combustion into the room itself, needs closer consideration when the ventilation is not very efficient.

Of the products of combustion of coal-gas water

vapour may in some cases be beneficial in raising the humidity of dry air; it may be harmful if the air is already very humid. Carbon dioxide is harmless in any concentration likely to occur; carbon monoxide is not found in appreciable amounts in the gases from well-designed appliances kept in good condition. The only products likely in the opinion of the committee to be harmful in themselves are the oxides of sulphur and of nitrogen, the former derived from combustion of sulphur in the gas, the latter always found when air is heated sufficiently for oxygen and nitrogen to combine. A trained nose will usually detect the higher oxides of nitrogen in the air of a room overheated and underventilated when gas is the fuel (incidentally it may be mentioned that the same thing must occur with many electric heaters); the oxides of sulphur are less apparent, but sulphur trioxide probably accounts for the acrid dry smell of such rooms. Sulphur dioxide, which is the first product of combustion of sulphur, is just detectable when its concentration reaches 2 parts per million (volumes), and may be harmful if 10 parts are present, although this is uncertain.

The committee conclude that discomfort from overheating would be experienced before harmful concentrations of sulphur and nitrogen oxides were reached and the gas would be turned out or lowered. This conclusion seems to be based on sound common sense. The effect on metal work in rooms where the products of combustion are freely diffused was not within the committee's reference. It would probably be considerable.

INFECTIOUS DISEASE

IN ENGLAND AND WALES DURING THE WEEK ENDED AUGUST 3RD, 1935

Notifications.—The following cases of infectious disease were notified during the week: Small-pox, 0; scarlet fever, 1664; diphtheria, 883; enteric fever, 61; acute pneumonia (primary or influenzal), 436; puerperal fever, 36; puerperal pyrexia, 104; cerebro-spinal fever, 22; acute poliomyelitis, 30; polio-encephalitis, 1; encephalitis lethargica, 4; dysentery, 20; ophthalmia neonatorum, 114. No case of cholera, plague or typhus fever was notified during the week.

The number of cases in the Infectious Hospitals of the London County Council on August 9th was 2653, which included: Scarlet fever, 776; diphtheria, 958; measles, 32; whooping-cough, 426; puerperal fever, 23 mothers (plus 5 babies); encephalitis lethargica, 269; poliomyelitis, 7. At St. Margaret's Hospital there were 22 babies (plus 11 mothers) with ophthalmic neonatorum.

Deaths.—In 121 great towns, including London, there was no death from small-pox or enteric fever. 10 (0) from measles, 6 (2) from scarlet fever, 11 (2), from whooping-cough, 27 (2) from diphtheria, 56 (10) from diarrhœa and enteritis under two years, and 5 (0) from influenza. The figures in parentheses are those for London itself.

The deaths from diarrhœa and enteritis are little more than last week, the highest numbers being Birmingham 8, Liverpool 7, Manchester 5, no other great town more than 2. Of the 9 fatal cases of whooping-cough outside London 2 occurred at Birmingham; the others were widely scattered. Liverpool reported 4 deaths from measles, Sunderland 2. No great town reported more than 2 deaths from diphtheria.

The number of stillbirths notified during the week was 248 (corresponding to a rate of 36 per 1000 total births), including 30 in London.

THE SERVICES

ROYAL NAVAL MEDICAL SERVICE

Surg. Capt. K. H. Hole, O.B.E., to *Drake* for R.N.B.

Surg. Comdr. C. H. Savory to *Pembroke* for R.N.B.

Surg. Lt. D. W. Pratt to *Weston* and G. D. Wedd to *Victory* for Haslar Hospital.

ROYAL NAVAL VOLUNTEER RESERVE

Surg. Lt. A. S. Pearson to *Victory* for R.N.B.

Surg. Sub-Lt. D. Shute to be Surg. Lt.

ROYAL ARMY MEDICAL CORPS

Maj. H. G. Hobson, O.B.E., M.C., retires on ret. pay.

REGULAR ARMY RESERVE OF OFFICERS

The undermentioned having attained the age limit of liability to recall, cease to belong to the Res. of Off.: Lt.-Col. R. J. C. Thompson, C.M.G., D.S.O., and Majs. A. W. Howlett and H. T. Stack, O.B.E.

Capt. H. L. Oldershaw resigns his commn.

ROYAL AIR FORCE

Flight Lt. F. W. P. Dixon, M.B.E., to R.A.F. Depôt, Uxbridge; R. G. Freeman to R.A.F. Hospital, Cranwell; and H. J. Melville to R.A.F. Station, Boscombe Down.

Flight Lt. G. E. Church is promoted to the rank of Squadron Leader.

Dental Branch.—Flight Lts. W. D. Guyler to R.A.F. Depôt, Uxbridge, and B. L. Harrington to Home Aircraft Depôt, Henlow.

INDIAN MEDICAL SERVICE

Capt. E. G. Hurd-Wood to be Maj. (provl.).

Lts. (on prob.) to be Capts. (on prob.): J. Brebner, W. J. Virgin, H. W. G. Staunton, J. D. Gray, D. W. Taylor, P. H. Addison, C. U. Wickham (since decd.), March 11th, 1935, with seniority Oct. 25th, 1934; S. G. O'Neill, F. J. Doherty, J. W. D. Goodall, and C. B. Miller.

The undermentioned officers retire: Lt.-Cols. A. N. Thomas, D.S.O., H. W. Acton, C.I.E., and A. P. G. Lorimer (on account of ill-health).

Capt. K. C. Virmani relinquishes his temp. commn.

The undermentioned officer has vacated appt. in India:—

D.A.D.M.S.—Lt.-Col. P. S. Tomlinson, M.C., R.A.M.C., and has been appointed as Deputy Director of Medical Organisation for War.

The undermentioned appts. have been made in India:—

D.A.D.M.S.—Lt.-Col. G. F. Allison, M.C., R.A.M.C.

D.A.D.P.—Maj. C. Scales, R.A.M.C.

COLONIAL MEDICAL SERVICE

Dr. A. C. de B. Helme has been appointed Medical Officer, Tanganyika, and Dr. D. E. C. Mekie, Medical Officer, Malaya. Dr. H. P. Fowler becomes Senior Health Officer, Gold Coast; Dr. P. E. M. Lanier, Senior Medical Officer, Seychelles; and Dr. S. M. Vassallo, Senior Specialist Officer, Zanzibar.

FELLOWSHIP OF MEDICINE AND POST-GRADUATE MEDICAL ASSOCIATION.—The next series of post-graduate courses will begin in September: heart and lung diseases at the Royal Chest Hospital (Mondays, Wednesdays, and Fridays, 8 P.M., from Sept. 9th to 27th); pathological and clinical demonstration at the National Temperance Hospital (8 P.M., Tuesdays and Thursdays, Sept. 10th to 26th); infants' diseases at the Infants Hospital (Sept. 2nd to 13th); medicine, surgery, and the specialties at the Westminster Hospital (Sept. 16th to 28th); chest diseases at the Brompton Hospital (Sept. 23rd to 28th), and ophthalmology at the Royal Westminster Ophthalmic Hospital (Sept. 28th and 29th). The courses at the Royal Chest and National Temperance Hospitals are primarily, but not exclusively, intended for candidates for the M.R.C.P. examination. Other forthcoming courses include: anatomy and physiology (primary F.R.C.S.) at the Infants Hospital (8 P.M., on Mondays, Wednesdays, and Fridays, from Oct. 7th to Nov. 29th); endocrinology at the National Temperance Hospital (8.30 P.M., on Tuesdays and Thursdays from Oct. 8th to 24th); and gynæcology at the Medical Society of London (4 P.M., on Thursdays from Oct. 10th to Nov. 7th). Detailed syllabuses of all courses can be obtained from the secretary of the Fellowship, 1, Wimpole-street, London, W.1, within a few weeks of the starting date of each course. Courses of instruction, arranged by the Fellowship, are open only to members and associates.

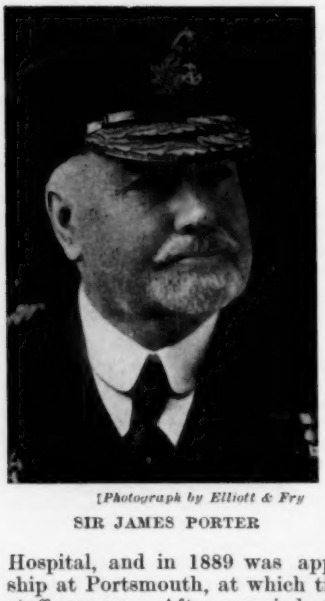
OBITUARY

SIR JAMES PORTER, M.D. Aberd., K.C.B., K.C.M.G.

LATE DIRECTOR-GENERAL, MEDICAL DEPARTMENT, R.N.

WE regret to announce the death on Friday, August 9th, of Surg. Vice-Admiral Sir James Porter, a distinguished former director-general of the medical department of the Royal Navy. The event occurred at St. John's Lodge, Wakefield, at the age of 83.

James Porter was born in 1851 and educated at the Old Grammar School, Aberdeen, and the University of Aberdeen, where he graduated in arts in 1874 and in medicine three years later. Immediately on medical qualification he entered the medical service of the Royal Navy. He was first appointed in 1877 to the Mediterranean fleet, but later joined the troopship, *Orontes*, in which he served throughout the Egyptian war of 1882, obtaining the medal and Khedive's Star. In 1884 and 1885, on duty with the Royal Marine Battalion sent to the Sudan, he was present at the action of Tofrek (March 22nd, 1885) and received two clasps to his Egyptian decoration for services during the campaign. Following a year's home service in Chatham he proceeded to Bermuda in medical charge of the Naval Hospital, and in 1889 was appointed to the depôt ship at Portsmouth, at which time he was promoted staff surgeon. After a period of service in the Mediterranean he returned to Bermuda, holding now the position of medical officer to the dockyard there; at the expiration of this appointment he became senior medical officer of the *Britannia* at Dartmouth. Here Porter in his relations with the cadets earned an enviable reputation both for ability and for the helpful and kindly attitude displayed towards his young charges; at the expiration of his time on the *Britannia* he was promoted fleet surgeon. In 1889 occurred the South African war, and Porter saw much service as fleet surgeon with the Naval Brigade, was mentioned in dispatches, obtained the Queen's medal with eight clasps, and was specially promoted to the then existing position of Deputy Inspector-General of Hospitals and Fleets.



[Photograph by Elliott & Fry

SIR JAMES PORTER

The record of his service becomes now one of steady well-merited success. In 1901 he went to Haslar as second in charge; in the following year he was in charge of the Royal Hospital at Chatham, when he received the C.B.; and in 1905 he held the same appointment at Gibraltar; he was promoted Inspector-General in 1906, becoming two years later the head of his branch of the profession as Medical Director-General of the Navy with the rank of Vice-Admiral. In 1910 he received the K.C.B., and the year before the European war had retired from the active list, but on the declaration of hostilities he returned to the Navy for special duties at the Admiralty arising out of his long and practical experience in the working of the naval medical service. During the European war he served as principal hospital transport officer in the Mediterranean and the Dardanelles, and in 1916 was appointed K.C.M.G., having also received the Star and Allies medal and the Victory medal. This is the brief story of a distinguished national servant.

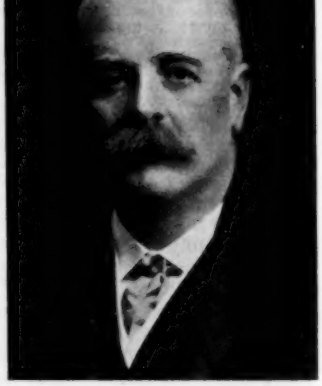
At different times in his career the quality of Porter's work was recognised without the limits of his particular service. He was honorary physician to the King, and a Knight of Grace of the Order of St. John of Jerusalem; his own university and the University of Edinburgh made him a LL.D., and he held the Coronation medal. He married in 1886 Emma, daughter of the late Inspector-General M. W. Cowan, R.N., by whom he had one daughter. He had been a widower for three years. He will be buried at sea.

WILLIAM THOMAS HOLMES SPICER, M.B. Camb., F.R.C.S.Eng.

CONSULTING OPHTHALMOLOGICAL SURGEON, ST. BARTHOLOMEW'S HOSPITAL

THE death occurred on August 8th at his house in Wimbledon of Mr. Holmes Spicer, the ophthalmologist, as well known as an organiser and an authority on the teaching of his science as he was as clinical exponent. He was in his seventy-fifth year.

Holmes Spicer was born at Saffron Walden and educated at the school there, and later at Barnet, whence he proceeded to Cambridge. At first attached to no college, in 1879 he entered Gonville and Caius, and graduated in 1880 with honours in the Natural Science Tripos. He proceeded for his medical education to St. Bartholomew's Hospital, took the diploma of M.R.C.S. in 1884, the Cambridge M.B. in 1886, and the F.R.C.S. Eng. in 1888. At St. Bartholomew's he had a prominent career, being Bentley surgical prizeman and later Brackenbury surgical scholar. He was house surgeon and ophthalmic house surgeon to the hospital, and followed many distinguished predecessors in being president of the Abernethian.



[Photograph by Lafayette

MR. HOLMES SPICER

After a short period of general practice he decided to follow the specialism in which he created for himself a successful position. Following a clinical assistancy at Moorfields he was appointed ophthalmic surgeon to the Metropolitan Hospital and to the Victoria Hospital for Children, and did noticeable work at the latter institution in connexion with scurvy rickets, making communications on the subject to

the Transactions of the Ophthalmological Society. In 1898 he was appointed surgeon to Moorfields, a position which he occupied for over 20 years; he was for a long period dean at the hospital, a post which he filled to admiration, the number of students mounting up as the organisation of the teaching, through Spicer and the staff, became perfected. In 1901 Bowater Vernon retired from his position as ophthalmic surgeon to St. Bartholomew's, and Walter Jessop succeeded him with Holmes Spicer as his junior colleague. Spicer was on the staff at St. Bartholomew's until 1920, when he retired under the age rule, but was appointed a governor of the institution and a member of the house committee in recognition of his valuable coöperation.

Spicer was a considerable author throughout the whole period of his hospital appointments, although he wrote no standard work on ophthalmology. He regularly contributed to the Transactions of the Ophthalmological Society, his articles covering a very large clinical area and displaying both physiological and practical wisdom. He wrote the article on the cornea in the Encyclopædia Medica, contributed to Posey and Wright's "Diseases of the Eye, Nose, Throat, and Ear," and edited the sixth edition of "Diseases of the Eye," by Nettleship, whom he had worked under and had succeeded at Moorfields. This edition appeared seven years after its predecessor. The original work dealt with the more common forms of ophthalmic disease and their characteristic symptoms, and laid down the broad principles of treatment, so that much revision might not be considered necessary, but Spicer greatly improved the work. In its new form descriptions of operations were simplified, statements not sanctioned by more mature experience were omitted, and new facts were added, making of the sixth edition of Nettleship's valuable manual a work thoroughly representing the state of ophthalmic knowledge at the day.

Spicer, who was twice married, leaves no children. In his leisure time, both while in practice and after retirement, Spicer painted, mainly in water-colour, and many may have seen his drawings privately or at exhibitions at the Royal Society of Medicine of work by medical artists. The portrait is reproduced from a photograph taken on his retirement from St. Bartholomew's.

Sir John Parsons writes: "The death of Mr. Holmes Spicer has bereft ophthalmologists of an esteemed colleague and a loyal friend. He brought to his work a sound knowledge of general science, acquired at that best of scientific schools, Cambridge; but in addition he was widely read in literature and history, and endowed with unusual artistic ability and outlook. Coming early under the influence of the most scientific teacher of his time, Edward Nettleship, any high flights of imagination and originality, of which he was fully capable, were coördinated and controlled by that stern but salutary taskmaster. With his gift of accurate draughtsmanship it was natural that his notes of cases should be profusely illustrated by sketches of abnormal conditions, often revealing to others the true nature of a case in a manner which verbal description alone failed to convey. He was specially interested in the innumerable types of corneal ulcer and opacity; and his Gifford Edmonds Prize Essay on 'Parenchymatous Keratitis,' &c., published as a monograph by the *British Journal of Ophthalmology*, embodied the work of a lifetime. It is enriched by many exquisite drawings and will ever remain a classical contribution to the subject. On holidays and in retirement he gave free rein to his artistic bent, revelling in the beauties of scenery and architecture, perpetuating many scenes with no mean skill. One such sketch, which he gave to me, has been highly praised by competent critics, and there is little doubt that, had he devoted his life to art, he would have attained a high position—but that would have been at the expense of his outstanding contributions to ophthalmology. It is difficult for one who is proud to claim the title of a friend to convey the charm of Spicer's personality. Of massive build and sedate demeanour, always unperturbed and somewhat slow of speech, he was full of kindliness and humour. It was a unique experience to travel abroad with him and witness his enjoyment of everything—including the 'good things' of life."

J. T. writes: "One who had been on terms of cordial and intimate friendship with him for more than 40 years may be permitted to add a few words to your tribute to Holmes Spicer. He was a big man physically, slow in his movements, but mentally very alert, with a quickness of observation which surprised one. It was a special pleasure to be on holiday with Spicer. The writer can look back on happy times in France, Italy, and Sicily with him and on many week-ends at Rye. The memory of these times is a very happy possession, and one can never forget the helpful and sympathetic care with which he conveyed back to London the friend suddenly seized with illness on one of those week-ends. Spicer's artistic gifts were much in evidence. His water-colours were his great pleasure, but in the writer's opinion his pencil sketches were even better, although he himself revelled in colours. He was very fond of music, and in early days singing was a pleasure to him and gave much pleasure to others. He was at his best among a few intimate friends when his genial presence, his keen sense of humour, and his inimitable reproduction of patients' tales combined to render an evening memorable."

WALTER GIFFORD NASH, F.R.C.S. Eng.

CONSULTING SURGEON, BEDFORD COUNTY HOSPITAL

THE death occurred on Friday, August 2nd, of Mr. Gifford Nash, one of the best-known provincial surgeons in the country and rightly recognised as a pioneer in more than one important direction of surgery.

Walter Gifford Nash was born in 1862 at the parsonage, Berden, Essex, the fourth son of the Rev. Frederick Gifford Nash. He was educated at the Gloucester Cathedral, and went for his professional education to the Middlesex Hospital, entering the medical school in 1882 with an entrance scholarship and other exhibitions. He took the English double qualifications in 1887, served as house physician, as house surgeon (under Henry Morris), and as obstetric house physician to the hospital, and in 1891, having been for a period house surgeon to the South Devon and East Cornwall Hospital, Plymouth, he obtained the F.R.C.S. Eng. In 1891 he went into practice in Bedford, much of his excellent work later being done in connexion with his appointment as surgeon to the Bedford County Hospital, although already he had made a mark by the original nature of contributions to this journal and elsewhere.

We owe to Mr. G. Gordon-Taylor the following summary of Gifford Nash's surgical career:—

"The death of Walter Gifford Nash has not only robbed Bedford and Bedfordshire of a great surgeon

and a charming personality, but British surgery is also the poorer for the loss of one who made notable contributions towards its advancement. It was always difficult to realise in conversation or in coöperation with Gifford Nash that he had been practising for nearly half a century, and that his earliest contribution to the columns of THE LANCET was made as long ago as June 21st, 1890, a number which also contained one of David Ferrier's Croonian lectures on cerebral localisation and the second Report of the Hyderabad Commission on chloroform.

"Gifford Nash commenced to practise when much of the surgery of the abdomen was still in its infancy. In 1887 Frederick Treves had performed the first deliberate appendicectomy, and Nash, embarking only two or three years later upon his own earliest appendicectomies, lacked the good fortune to have actually witnessed the operation carried out by another surgeon. It was not otherwise with gastric surgery; for Nash's earliest operations on the stomach, performed in the early nineties, were not modelled on anything observed in the theatres of other operators; he had to direct his own skilful hands. He was not long behind Sir Arbuthnot Lane and Sir Charles Ballance in successfully operating for infective thrombosis of the lateral sinus and ligaturing the internal jugular vein for this condition. One of his earliest contributions to surgical literature dealt with torsion of the undescended testicle, and although Nicoladoni had drawn attention to this complication in a foreign journal in 1884, Nash was the first British surgeon to describe the condition in a short paper in THE LANCET of June 6th, 1891. Not only can priority among British surgeons be claimed for him in recording this complication of the undescended testis, but he later recorded cases in which he was actually able by manipulation to successfully untwist the torsion, while he made other contributions to the literature of this subject. He seems to have had almost a flair for encountering the unusual in the surgery of that now-distant period, and his writings refer to such variegated activities as a successful nephrectomy for a pyonephrosis of the left half of a horseshoe kidney in an infant of 16 months, and the successful removal of a torsion of the testis in an infant 9 days old; he dealt successfully with a case of acute pancreatitis only five years after Fitz's classical description of that disease in 1889, and also with such a rarity as a primary hyperplastic tuberculosis of the stomach and duodenum.

"While his house surgeoncy to Sir Henry Morris in 1887 naturally led to a sustained interest in the problems of renal surgery, his surgical apprenticeship within the walls of Middlesex Hospital, when Bland Sutton was unravelling the pathology of ectopic gestation and indicating its surgical treatment, doubtless stimulated his enthusiasm for the surgery of this condition and may have been responsible for the numerous papers on this subject that frequently came from his pen. In the problems of cancer he also took a close interest. In 1902, as president of the South Midland Branch of the British Medical Association, he delivered an address containing much of interest in connexion with 'cancer houses,' 'dual cancer,' and other points dealing with the ætiological aspect of the disease.

"Nash's wealth of surgical experience was immense, and any appeal would always result in his unburdening himself of an abundance of information germane to the question at issue and of value to the interrogator who sought his advice and aid. He had capable hands and profound judgment; he inspired confidence everywhere, no less in the houses of the rich than in the cottages of the poor; he radiated enthusiasm amongst those who were his colleagues in the work of his life."

The interests of Gifford Nash were very wide and in all of them his energy and enthusiasm were displayed. In connexion with the Bedford hospital the County Guild was his conception and he was vice-president from its foundation nearly 30 years ago. He took a deep interest in the successful development of the Workers' Hospital Fund, another movement where he functioned as vice-president, and he was chairman of the convalescent home committee from its inception to the end of his life. In the development of the paying wards and the maternity department of the hospital he took a keen interest. Mr. J. A. Whitchurch, chairman of the managing board, in paying tribute to his activities, said that in the early years of the paying wards nearly two-thirds of the patients were under Gifford Nash's care, closing with the words, "For the last 23 years one of my most treasured experiences is the friendship, help, and advice of one I honoured and loved, a great surgeon, and a great gentleman, Walter Gifford Nash."

Mr. Gifford Nash married Miss Catherine Moore Wilson, daughter of the late Mr. Walter Wilson, of Plymouth, who survives him with six children, among whom are Mr. F. W. G. Nash, F.R.C.S., and Captain H. K. G. Nash, R.A.M.C.

ARTHUR SHILLITOE, M.B., B.Chir. Camb., F.R.C.S. Eng.

THE death has been announced of Arthur Shillitoe a well-known authority both on syphilis and life assurance problems. Born in 1860, he was educated at Cambridge, where he graduated as M.B., B.Chir. in 1894. He went for his clinical training to Guy's Hospital, and took the English double qualification in 1893, proceeding to the F.R.C.S. Eng. two years later. At Guy's he acted as assistant demonstrator of anatomy, and his first appointment showed the direction which his work would take and which was hereditary, for he became assistant surgeon to the London Lock Hospital and surgical registrar. While at the hospital he made many communications to medical societies and to the medical press, notably an investigation of a series of cases of pseudo-chancre which was published in these columns on May 30th, 1896. This communication made while Shillitoe was registrar at the Lock Hospital was mainly founded on observations made by his father in private practice, and the information as to recurring chancres supplied in this

[*Photograph by Elliott & Fry*

MR. SHILLITOE

paper was of a valuable nature, pointing to the elucidation of frequent difficulties in diagnosis. Later he collaborated in the production of D'Arcy Power and J. K. Murphy's "System of Syphilis," to which he contributed the section dealing with the primary lesions and early secondary symptoms of syphilis as seen in the female. In 1904 we published an article by Sir Arthur Keith, then lecturer on anatomy at the medical school of the London Hospital, and Shillitoe on the preputial glands of man, in which the embryological side of the subject was fully dealt with. Shillitoe was medical officer to the Atlas Insurance Company, and out of his experience arrived at certain conclusions on the relation of syphilis to life assurance which he published in the Transactions of the Life Assurance Medical Officers' Association in 1906. Thus in many directions he furnished the profession with practical knowledge in circumstances where exact diagnosis was of the first value.

Shillitoe had retired from active work for some time, and his death occurred suddenly in a Plymouth nursing-home on July 22nd.

DR. AMREIN

THE death in Zurich on August 2nd of Dr. Otto Amrein will be noted with regret by all who are interested in the climatic treatment of pulmonary tuberculosis. He was 62 and suffered from cardiosclerosis. Amrein's life coincided with the rise and decline of Arosa as an invalid resort. When he settled there shortly after qualifying in medicine at Zurich Jacobi's big German sanatorium had already been going for some years, and Dr. Roemisch, who is still living in retirement, was seeing most of the private patients who had begun to flock to the village of Arosa as an alternative to the more sophisticated town of Davos. Rüedi had taken with him a considerable clientele when he left Davos for Arosa, but did not long survive to enjoy it. Amrein's success was assured from the start; he was a first-rate clinician, cautious and sagacious, and with the personal qualities that retain the patient's confidence through months or years of monotonous life—for it must be remembered that sanatorium régime in the early years of the century was simply the application of hygienic-dietetic principles with no daily diversions such as artificial pneumothorax or gold injections. For twenty years or so Amrein was content with supervising his large practice in hotels and pensions, over the management of which his position enabled him to exert a controlling influence; but in 1914 was opened the sanatorium Altein in which was embodied every item of comfort and equipment designed for the kind of life he wished his patients to live. Circumstances combined to prevent it ever becoming the success he hoped. The death of a prominent financier, when it was half built, deprived it of the architectural features which would have made it a joy to behold as well as to live in, and the war gradually stemmed the flow of foreign patients, although later on the Red Cross was glad to find such a chance for its invalided officers. As the economic situation in Europe darkened one sanatorium after another in Arosa had to close its doors for patients, opening only for a month or two in the winter for sport. Through all these vicissitudes Amrein was the daily guide and counsellor of hundreds of phthisical English patients, many of whom owe him the health they now enjoy. He was always on the side of prolonged bodily rest, although he learnt that it was possible to make exceptions for the Anglo-Saxon temperament. He was immensely respected and a little feared by his colleagues, and consulted by the local authorities in every matter touching the place as a health resort. His familiar figure, with the slight limp which never interfered with his activities, will be much missed. If he had not been a doctor Amrein could have easily made his mark as a musician, for he was a pupil of the great Joachim, and continued throughout his life to take part in charitable concerts.

PHILIP JOHN LE RICHE, M.R.C.S. Eng.

THE death occurred on July 27th of Dr. P. J. Le Riche, who had been in practice before his retirement for some 30 years in Worthing. He was born at St. Heliers, Jersey, in 1863, the son of Mr. F. J. Le Riche, and he was educated at the Victoria College, Jersey, and University College, London, receiving also some of his medical training at Guy's Hospital. His first bent was towards ophthalmology, and he acted as an ophthalmological assistant at Moorfields, but later he made several voyages as surgeon to a steamship line. He then set up in practice at Worthing, and at the outbreak of war was in charge of The Grange auxiliary hospital, Worthing, and held also a captain's commission in the 4th Battalion, the Royal Sussex Regiment (T.A.). After the war he continued to practise in Worthing and became widely known both as a doctor and for various other activities. He was a useful local cricketer, was prominent in masonic circles, and under the name of "Kish" wrote a book called "Creation's Dawn," which attracted considerable public attention. Dr. Le Riche had for many years been a widower.

HOSPITAL COÖRDINATION IN LONDON

THE London Voluntary Hospitals Committee and the London regional committee of the British Hospitals Association have amalgamated under the title The Voluntary Hospitals Committee, County of London. The new committee represents both the governing bodies and the medical staffs of the 108 voluntary hospitals in the county of London, and will continue the consultations which have been taking place between the L.C.C. and the Voluntary Hospitals Committee since 1930, under Section 13 of the Local Government Act, 1929. A subcommittee has been appointed to meet representatives of the hospitals and medical services committee, L.C.C. The new committee will be an integral part of the B.H.A., and its functions will include the consideration of all subjects which may affect the voluntary hospitals within the county. The honorary officers are: chairman, Mr. Oliver N. Chadwyck-Healey; vice-chairman, Dr. A. M. H. Gray; treasurer, Sir Herbert Samuelson; secretary, Prof. Leonard S. Dudgeon.

KING GEORGE HOSPITAL, ILFORD.—Figures issued by the King Edward Hospital Fund for London show that out of a group of twelve hospitals with from 12 to 200 beds, this hospital was the only one with no available beds vacant, and the cost of maintenance per bed is less than the average. Income and expenditure for the first half of the year show the total income to be £14,000, as compared with £12,593 in the first half of 1934, and the expenditure was £18,509, an increase of £2962.

CORRESPONDENCE

TOXICITY OF AVERTIN

To the Editor of THE LANCET

SIR,—The report by Dr. Cole in your issue of August 3rd, and the letter from Sir Francis Shipway in your last issue, prompt me to record an example of the value of avertin. In August, 1932, two boys, brothers, aged 12 and 9, were admitted to the Dorset County Hospital suffering from meningitis, the organism of which was stated by the pathologist to be Pfeiffer's bacillus. Both boys were treated with continuous avertin anæsthesia, and apart from the initial lumbar puncture for diagnostic purposes, no other treatment was adopted. Full doses of avertin were given, three or four times in each 24 hours. These would cause the children to sleep for four or five hours; the head retraction disappeared, and there was no crying. It was possible to feed the children while they were coming round from the anæsthesia, though, in addition, fluids were given by the rectum. When the anæsthesia passed off, the heads of the children would retract, and they would begin to cry, and occasionally to scream.

After four days the younger child recovered sufficiently to stop the avertin, and in a week we were able to do the same with the brother. Recovery was complete, except for deafness in the older child, which persisted for a further week, and then cleared up completely.—I am, Sir, yours faithfully,

R. E. HOPE SIMPSON.

Beaminster, Dorset, August 9th.

PROCTALGIA FUGAX

To the Editor of THE LANCET

SIR,—Dr. Thaysen's article has served to arouse my interest in two cases which have shown symptoms similar to those he describes. In both cases the following features were common:—

1. The attacks were of uniform severity and of constant duration, each attack lasting about 5 minutes.
2. The pain was gnawing and cramp-like in character, and was neither preceded by nor followed by tenesmus or flatus.
3. The pain was localised in the rectal region immediately above the sphincter ani.
4. The attacks disappeared spontaneously and were followed by feelings of exhaustion and lassitude, and in both cases resulted in the development of an anxiety state. They were unaccompanied by any disturbance of intestinal function.
5. Rectal examination digitally and per sigmoidoscope failed to reveal any causation for the attacks, and during an attack there was no spasm of the anal region.
6. No method of treatment was of any avail.

In both these cases the attacks came on within a period of 30 minutes following masturbation, and depended, I suggest, upon venous engorgement following sexual excitement.

I am, Sir, yours faithfully,

WILLIAM BLYTH.

The Mental Hospital, Riccartsbar, Paisley, August 10th.

To the Editor of THE LANCET

SIR,—As a sufferer from this complaint for a quarter of a century may a humble member of the profession venture to offer an opinion on what seems to puzzle his betters? I first experienced the affection after rashly leaving off my natural-wool underclothing in a bleak April spell. Since then I can always tell the prevalence of an east wind by my own feelings and without looking at the weather. As to treatment, I have always got relief from guaiacum and sulphur in tablet form, but whether it is the sulphur or the guaiacum that is the specific I cannot say. What I do claim is that the trouble is a neuritis, and I have read in Hilton's "Rest and Pain" (I cannot at the moment find the reference) that there is no less reason why the rectum should be subject to neuritis than any other part of the body. At first I feared *everything* including cancer as the cause and consulted a surgeon, but he could find nothing amiss locally, and anyhow I am still alive!

I am, Sir, yours faithfully,

August 9th. A SEPTUAGENARIAN.

PRURITUS

To the Editor of THE LANCET

SIR,—I have read and appreciated Lord Horder's address printed in your last issue. My own view is that X radiation should be regarded as a useful method of desensitisation in these cases. As Lord Horder has so aptly stressed, it is the underlying general ætiological factors which must be discovered and remedied. Failure in this direction will inevitably lead to a recurrence of the pruritus when further desensitising doses of X rays will give at best but temporary relief. In my experience cure may be effected in more than 50 per cent. of cases, and I do not believe "heavy doses" to be ever necessary.

I am, Sir, yours faithfully,

Putney, S.W., August 13th. WARNER COLLINS.

SIGNIFICANCE OF PERSISTENT HIGH BLOOD PRESSURE

To the Editor of THE LANCET

SIR,—Dr. L. N. Jackson's article in your issue of August 10th is most interesting, but I should like to join issue with him on one point. He states that patients with essential hypertension do badly on insulin. Surely this is too sweeping? Indeed diabetic accumulations in the blood probably contribute to the production of arterio-sclerosis.

I have now under my care 11 diabetics who are the subjects of persistent high blood pressure and have taken insulin for periods varying from eight months to six years with no ill-effects. All are over 55 years of age, and four are over 70. The systolic blood pressures range from 180 to 280 mm. Hg and the dose of insulin from 10 to 60 units a day. One patient has been able to leave off insulin owing to increased tolerance for sugar, and two have reduced it.

I believe that diabetics with high blood pressure derive nothing but benefit from insulin. Whether hypoglycæmia is harmful is another question. Joslin and others consider it dangerous in cases of angina pectoris and arterio-sclerosis, and it is reasonable to suppose that a reduction of the sugar content of blood passing through narrowed coronary arteries may fatally starve the heart. In my own patients with hypertension, the lowest point found during routine examination of the fasting blood-sugar was 0·08 per cent.—on the border line of hypoglycæmia—but one patient had an attack of hypoglycæmic coma when on holiday, and returned home apparently none the worse.

It seems unjustifiable to withhold insulin from diabetics with high blood pressure, either on theoretical or practical grounds; although when there is reason to suspect coronary arterio-sclerosis it is well to avoid hypoglycæmia.

I am, Sir, yours faithfully,

Hove, August 13th. G. M. WAUCHOPE.

POOLED HUMAN MILK FOR INFANT FEEDING

To the Editor of THE LANCET

SIR,—In your annotation last week on pooled human milk for use in the feeding of premature infants and for babies suffering from alimentary disturbances, you report the doubt that Prof. W. Catel of Leipzig has thrown on its value. He has found that the mortality and morbidity of prematures is higher when the breast milk given is sterilised by heating than when it is given fresh. By this and other work he has been led to challenge the value of sterilised human milk.

It would be a pity to leave the matter at this point. The lesson to be learned from his observations is that sterilisation of breast milk by heating results in a reduction of those antibodies on which much of the comparatively high (passive) immunity of breast-fed infants depends. This is exactly what one would expect. Clearly the problem is to know how pooled human milk can best be stored so that deterioration does not occur. Some of your readers may like to know that breast milk, after being collected aseptically, may be stored at −10° C. in a refrigerator. In centres where this method is followed the milk, frozen solid, seems to keep indefinitely, and satisfactory results are obtainable by its use. This does not diminish the value of Prof. Catel's comparison between heated and fresh breast milk; indeed the evidence he reports of the deleterious effect of sterilisation by heating gives force to the argument in favour of low-temperature storage.

I am, Sir, yours faithfully,

REGINALD LIGHTWOOD.

Brook-street, W., August 13th.

DR. GRIFFITH EVANS

ON HIS HUNDREDTH BIRTHDAY

IN celebrating the centenaries of distinguished medical men we are seldom able to enjoy their presence among the celebrants. The hundredth birthday of Dr. Griffith Evans therefore gives peculiar pleasure to his colleagues and successors, and they are glad to recognise his services in two branches of medical work, for he is not only a graduate of medicine but also, and primarily, the Father of the Veterinary Profession in this country.

Dr. Evans was born at Towyn in Merioneth on August 7th, 1835, and qualified from the Royal Veterinary College at the age of twenty. He obtained a commission in the Army in 1860, and while stationed at Montreal paid a memorable visit to the armies of the North in the American Civil War, receiving his authorisation in a personal interview with Lincoln. The thesis which gave him his medical degree at McGill University in 1864 is said to have recognised the infectious nature of tuberculosis and emphasised the desirability of open-air treatment. Service in India followed, and in 1880, when he was inspecting veterinary surgeon to the Government of Madras, he made the investigations on surra which have won him lasting repute. Surra is a troublesome disease of horses, mules, camels, and cattle, occurring in India, Burma, Indo-China, and North Africa, and in discovering the causal organism Evans was also demonstrating, for the first time, that trypanosomes are capable of causing illness. He showed that the *Trypanosoma evansi* of surra is transferable by subcutaneous injection and by passage into the stomach, and his observations were permanently commemorated when Steel gave the organism its present designation. The other species or variety which bears his name is *Filaria sanguinis evansi*, isolated in 1882 from the blood of camels.

Dr. Evans retired from the Army in 1890, and before going to Bangor lived for some years in Cardiff where his daughter, Dr. Erie Evans, was in practice, and where he later renewed old acquaintance with Sir William Osler. His services to veterinary medicine have at various times been recognised by the conferment of the honorary degree of D.Sc. by the University of Wales, the Steel medal of the Royal College of Veterinary Surgeons, the Mary Kingsley medal of the University of Liverpool, and the freedom of Bangor.

On his hundredth birthday Dr. Evans was confined to his room, for he has lately fractured both thighs; but he was able to entertain a series of visitors bearing messages of congratulation. First among them were members of his own family, including Dr. Evans and Mrs. Garfitt (daughters), Miss Garfitt (niece), Mr. and Mrs. G. H. Jones (son-in-law and daughter), Mr. Evans (son), and Mr. Bevan Jones (grandson). Sir Frederick Hobday, Captain W. H. Savage, and Mr. W. T. Rowlands represented the veterinary profession, and Sir Frederick brought a congratulatory scroll from the Royal Veterinary College signed by the Duke of Gloucester as president and himself as principal. In accepting this scroll Dr. Evans expressed his gratitude at receiving it from the College where he began his professional career and spoke of the benefits he had received from his teachers there—notably Spooner, Simmons, and Morton. Recalling his early adventures in microscopy he told how he had made slides of blood—his own and that of animals—under different conditions of fasting and digestion. "I was the youngest student at College," he said, "and I was a ruminating animal!"

Telegrams were then delivered from the King and Queen, the Prince of Wales, the Royal College of Veterinary Surgeons, the Liverpool School of Tropical Medicine, the Welsh National School of Medicine, and other bodies. The mayor and town clerk of Bangor brought an address from the city council, and Dr. Evans warmly thanked them for expressing the friendship of "the people amongst whom I have lived." A message from Mr. Lloyd George, his fellow freeman of Bangor, referred to the pride felt by the Principality in Dr. Evans's lifework.

WHITE STICKS FOR BLIND PERSONS.—Just before the House of Commons rose Sir IAN FRASER asked the Minister of Transport if he was aware that more than 4000 blind persons have purchased or been given white walking-sticks for use when walking alone to indicate to motorists that they were blind; and if he would help to make the significance of these white sticks more widely known among the motoring public by giving instructions that a query with regard to them should be included in the driving tests now being given, and by including a reference to them in any future editions of the highway code or other propaganda which he might issue.—Mr. HORE-BELISHA replied: I have tried to assist the object which my hon. friend has in mind, and I have let it be known that the carrying of white sticks by the blind has my approval and support. I am always ready to advance the project to which my hon. friend refers by any means open to me and it will certainly be relevant to consider, when the highway code comes to be revised, whether it would be appropriate to include advice on this subject. On the other hand, it should be borne in mind that there are certain possible objections to this course; for example, blind people might rely too much on their sticks, the sticks might be used by people who are not blind, and people who are blind or nearly so and did not wish to use the sticks might be prejudiced in the event of an accident.

MEDICAL NEWS

Westminster Hospital Medical School

The annual dinner of old students of this school will be held on Saturday, Sept. 28th, at the Trocadero Restaurant, Shaftesbury-avenue, London, W., when the chairman will be Dr. Vivian Orr. On Sept. 28th and 29th a morning course of lectures open to all medical practitioners will be held at the school. On Monday, Sept. 30th, Major Ian Hay Beith will deliver the inaugural address at the Caxton Hall, S.W. He will speak on the Privileged Profession.

Society for Provision of Birth Control Clinics

A lecture on the theory and practice of contraception will be given by Dr. Gladys Cox on Friday, Sept. 13th, at the Walworth Women's Welfare Centre, at 6 P.M. At the centre demonstrations will be given at 6 P.M. and at 7 P.M. on Friday, Sept. 20th, and at the same hours on Friday, Sept. 27th. The fee for the lectures and demonstrations at the centre is 5*s.*; they are open to medical practitioners and to students who have completed their gynæcological course, and those wishing to be present are asked to signify at which of the alternative hours they wish to attend. The address of the society is 153A, East-street, Walworth-road, London, S.E.17.

Post-graduate Courses in Berlin

An introductory course on homœopathy will be held from Oct. 14th to Nov. 9th by the Berlin Academy for Post-Graduate Medical Study. Other courses will include diseases of the throat, nose, and ear (Sept. 30th–Oct. 12th); infectious disease (Sept. 30th–Oct. 5th); internal medicine (Oct. 7th–19th); eugenics (Oct. 7th–12th); tuberculosis (Oct. 21st–26th); and surgical treatment of intrathoracic diseases with special reference to pulmonary tuberculosis (Oct. 28th–Nov. 1st). Special courses in all branches of medicine, with practical work, are held every month. Further information may be had from the Academy at Robert Koch-Platz 7 (Kaiserin Friedrich Haus), Berlin, N.W.7.

King's College Hospital

Mrs. Kate Madders, M.B., has been awarded a Halley Stewart fellowship in order to continue investigations under the direction of Dr. R. D. Lawrence in the diabetic clinic of the hospital.

Bromley Hospital

The building of the £35,000 extension at this hospital has now begun. Two new wards are being added, a massage department provided, and the nurses' home extended.

Three Counties Hospital, Arlesey

A new nurses' home to house 102 nurses is to be built on the hospital estate. The estimated cost is over £24,000, and the three counties Bedfordshire, Hertfordshire, and Huntingdonshire have been asked to approve the scheme so that building may begin before the winter.

Crewe Memorial Hospital

The foundation-stone of a new women's ward has been laid at this hospital. The ward will complete the extensions embarked upon a few years ago. Last year 784 patients were treated and the cost of maintenance was over £5000.

North Devon Infirmary

There has been a large increase in expenditure at this institution, mainly due to alterations and repairs, but the building is now in a good state of repair. Ordinary income amounted to £5365, a decrease of £278. Patients' payments were down by £269, and contributory schemes showed an increase of £265.

Derbyshire Royal Infirmary

In less than fifteen months £43,000 has been subscribed towards the £90,000 needed for the new ward block at this hospital.

Manchester public health authority has decided to organise a joint advisory board for the purpose of securing greater coördination and efficiency among the hospitals in the area.

Mr. Leslie Paton has been elected an honorary member of the American Medical Association.

Tewkesbury's New Hospital

This hospital, which has been built at a cost of about £9000, was opened recently.

Putney Hospital for Incurables

The Duchess of York has opened a new nurses' home at this hospital. The home has been needed for some time but funds were lacking. About £10,000 is still wanted.

Hull Royal Infirmary

This institution has had a deficit for six successive years and the accumulated debit balance is now over £28,000. Annual subscriptions and donations are lower than they were five years ago, while the infirmary's accommodation and expenditure have increased by 35 per cent.

Cardiff Royal Infirmary

A ward has been named after Sir William Diamond, the chairman of this institution, in recognition of his 15 years' service to it. When he became chairman the bank overdraft was £70,000, which has since been wiped out.

Sequel to a Pilgrimage

Thirteen cases of enteric fever are reported from Glasgow and neighbouring counties about three weeks after the return of pilgrims from Lourdes, and it is suggested that infection occurred somewhere on the journey. A case at the Fazackerley Hospital, Liverpool, is also attributed to the same journey.

Gallipoli and Salonika Revisited

Those who served in the Near East during the late war will be interested to learn that a reunion pilgrimage cruise to Gallipoli and Salonika has been arranged by a committee of ex-Service men. Those taking part will leave Liverpool in the *Lancastria* on May 1st, 1936, returning on the 23rd, and calls will be made at Malta, Gallipoli, Salonika, and Istanbul. Particulars may be had from the Joint Hon. Secretaries, Gallipoli and Salonika Pilgrimage Committee, 3, Shaftesbury-avenue, Chesterfield. Early application is advised.

AN EDINBURGH LABORATORY

IN his latest annual report as curator Sir Robert Philip shows that valuable work is continuously in progress at the laboratory of the Royal College of Physicians of Edinburgh. Over 15,000 pathological and biochemical reports were issued in 1934, including 3468 histological reports most of which were for the determination of malignant growth. Of late years the reports of tumour growths have been collected for investigation, and a "cabinet gallery of tumours" has been formed which includes some notable special collections—for example of bone sarcomata and tumours of the brain. This collection has been of special value to the Cancer Control Organisation for Edinburgh and the South-East of Scotland, and specimens of special tumours have been sent to several centres both in this country and abroad. The Cullen prize has been divided between Lieut.-Colonel A. G. McKendrick, superintendent of the laboratory, and Sir Frederick Menzies, medical officer of health for the county of London, and the Lister fellowship has been awarded to Dr. Edith K. Dawson for her work on the pathology of the breast. The medical profession in Edinburgh are exceptionally fortunate in having this excellent laboratory at their disposal.

Appointments

CRAIG, N. S., M.D. Manch., has been appointed Medical Superintendent, St. Peter's Hospital, L.C.C.

GILL, W. J., M.D. Aberd., F.R.C.S. Edin., Medical Superintendent, Paddington Hospital, L.C.C.

Vacancies

For further information refer to the advertisement columns

Albert Dock Hospital, Connaught-road, E.—Res. M.O. At rate of £110.

Ashford Hospital, Kent.—H.S. £150.

Aylesbury, Royal Buckinghamshire Hospital.—Second Res. M.O. At rate of £150.

Barnsley County Borough.—M.O.H., &c. £900.

Belfast, Royal Maternity Hospital.—Two Res. H.S.'s. At rate of £100 and £52 respectively.

Birkenhead Municipal Hospital.—Sen. Res. M.O. £350. Also Res. M.O. £300.

Birmingham Education Committee.—Asst. School M.O. £500.

Birmingham, Little Bromwich Hospital for Infectious Diseases.—Jun. Res. M.O. £300.

Birmingham and Midland Hospital for Women.—H.S. At rate of £100.

Board of Control, Rampton State Institution for Mental Defectives, nr. Retford.—Deputy Med. Supt. £847. M.O. £515.

Bolton Royal Infirmary.—Res. Surg. O., also Asst. Res. Surg. O. £250 and £200 respectively.

Bootle General Hospital.—H.S. to Spec. Dept. At rate of £150.

Bradford Royal Eye and Ear Hospital.—H.S. £160.

Bristol, City and County of.—Asst. M.O.H. £500.

British Postgraduate Medical School, Ducane-road, Shepherd's Bush, W.—Cas. O.'s. At rate of £150. Also H.P.'s and H.S.'s.

Burton-on-Trent General Infirmary.—H.S. £150.

Central London Ophthalmic Hospital, Judd-street, St. Pancras, W.C.—Sen. and Jun. H.S.'s. £120 and £100 respectively.

Coventry and Warwickshire Hospital.—Res. Surg. O. £200.

Denbigh, North Wales Sanatorium.—Asst. Tuberculosis M.O. £500.

Denbighshire County Council.—County M.O.H., &c. £1100.

Dewsbury and District General Infirmary.—Sen. H.S. £200.

Doncaster Royal Infirmary.—H.S. to Eye and Ear, Nose, and Throat Depts. £175.

Dreadnought Hospital, Greenwich, S.E.—Receiving Room Officer. Also H.P. At rate of £200 and £110 respectively.

Dumfries and Galloway Royal Infirmary.—Supt. £500.

East Ham Memorial Hospital.—Pathologist. £300.

Epsom County Hospital.—Res. Asst. M.O. At rate of £250.

French Hospital and Dispensary, 172, Shaftesbury Avenue, W.C. Jun. Res. M.O. £100.

Gordon Hospital for Rectal Diseases, Vauxhall Bridge-road, S.W. Hon. Anæsthetist. 20 guineas.

Guildford, Warren-road Hospital.—Res. Asst. M.O. At rate of £250.

Harrogate and District General Hospital.—H.P. and Cas. O. Also H.S. Each at rate of £150.

Huddersfield, Mill Hill Isolation Hospital.—Res. M.O. £350.

Huddersfield, Storthes Hall Mental Hospital, Kirkburton.—Fifth Asst. M.O. £350.

Hull, Anlaby-road Institution.—Asst. M.O. £350.

Hull Royal Infirmary.—Third H.S. At rate of £150.

Iraq Government.—M.O. Dinars 70 a month.

Kingston and District Hospital.—Visiting Physician. £300. Also Visiting Aural S., Orthopædic S., and Neurologist.

Leamington Spa, Warneford General Hospital.—Res. H.S. to Cas., &c., Dept. At rate of £150.

Leicester City Mental Hospital, Humberstone.—Third Asst. Res. M.O. £350.

Leicester County Sanatorium and Isolation Hospital, Markfield.—Res. M.O. £400.

Leicester Royal Infirmary.—H.S.'s, Cas. O., H.P.'s, Jun. Res. Anæsthetist. Each at rate of £125. Also Jun. Cas. O. At rate of £100.

Lincoln, The Lawn.—Asst. M.O. £300.

Liverpool, Alder Hey Hospital.—Res. Asst. M.O. £200.

Liverpool, City Infectious Diseases Hospitals.—Asst. Res. M.O. £200.

Liverpool Heart Hospital.—H.P. At rate of £100.

Macclesfield General Infirmary.—Second H.S. £150.

Manchester, Booth Hall Hospital.—Two Jun. Asst. M.O.'s. Each at rate of £200.

Manchester Hospital for Consumption, &c.—Res. M.O. for Ear, Nose, and Throat Dept., St. Anne's Home, Bowdon. £200.

Manchester, Royal Children's Hospital, Gartside-street.—Two Asst. M.O.'s. Each at rate of £150.

Marie Curie Hospital, 2, Fitzjohn's-avenue, N.W.—Pathologist. £650.

Middlesbrough, North Riding Infirmary.—Third H.S. £125.

Miller General Hospital, Greenwich-road, S.E.—2 Cas. O.'s. Each at rate of £150. H.P. and H.S. Each at rate of £100.

Nelson Hospital, Merton, S.W.—Two Res. H.S.'s. Each at rate of £100.

Newcastle-upon-Tyne, Hospital for Sick Children.—H.P. or H.S. At rate of £100.

Northampton General Hospital.—H.S. At rate of £150.

Northumberland County.—Sen. Tuberculosis Officer. £800.

Norwich, Jenny Lind Hospital for Children.—Res. M.O. £120.

Norwich, Norfolk and Norwich Hospital.—H.S. to Surg. Outpatient Dept. £200.

Nottingham General Hospital.—H.S. to Ear, Nose, and Throat Dept. At rate of £150.

Peterborough and District Memorial Hospital.—Pathologist. £500.

Plymouth, City General Hospital.—Asst. M.O. Also Jun. Asst. M.O. £300 and £250 respectively.

Plymouth, Prince of Wales's Hospital.—H.P. and H.S. Each at rate of £120.

Portsmouth Royal Hospital.—H.P. At rate of £130.

Preston and County of Lancaster Royal Infirmary.—Two H.S.'s. At rate of £250 and £150 respectively.

Prince of Wales's General Hospital, N.—Hon. Surg. Reg. to Ear, Nose, and Throat Dept. £100. Also Jun. H.P. and two Jun. H.S.'s. Each at rate of £90.

Princess Louise Kensington Hospital for Children, St. Quintin-avenue, Kensington, W.—H.P. At rate of £100.

Reading, County Borough, Battle Hospital.—Res. Asst. M.O. £300.

Richmond, Surrey, Royal Hospital.—Jun. H.S. At rate of £100.

Rotherham County Borough Education Committee.—Sen. School M.O. £750.

Rotherham Hospital.—Cas. H.S. £150.

Royal Chest Hospital, City-road, E.C.—Med. Reg. £50.

Royal Eye Hospital, St. George's-circus, Southwark, S.E.—H.S. Also Asst. H.S. At rate of £150 and £100 respectively.

Royal Northern Hospital, Holloway, N.—H.P. Also Obstet. H.S. Each at rate of £70.

Ruthin Castle, N. Wales.—Jun. Res. Physician. £200.

St. Helens County Borough.—Asst. M.O.H. £500.

St. Leonards-on-Sea, Buchanan Hospital.—Jun. H.S. £125.

St. Paul's Hospital for Diseases of the Genito-Urinary Organs, &c. Part-time Pathologist.

Sheffield Royal Infirmary.—Second Asst. Cas. O. At rate of £80.

South-Eastern Hospital for Children, Sydenham, S.E.—Two Res. M.O.'s. Each at rate of £100. Also Hon. Pathologist. £50.

South London Hospital for Women, Clapham Common, S.W.—H.P. At rate of £100.

Tilbury Hospital, Essex.—H.S. At rate of £140.

Tunbridge Wells, Kent and Sussex Hospital.—H.S. to Ear, Nose, and Throat and Ophthalmic Depts. £150.

University of London.—Graham Scholarship in Pathology. £300.

Ventnor, Isle of Wight, Royal National Hospital for Consumption, &c.—Jun. Asst. Res. M.O. At rate of £300.

Weir Hospital, Grove-road, Balham, S.W.—Sen. Res. M.O. £200. Jun. Res. M.O. £150.

West London Hospital, Hammersmith-road, W.—H.P. and two H.S.'s. Each at rate of £100.

Wimbledon Hospital, Thurstan-road, S.W.—Res. M.O. At rate of £150.

Wolverhampton, Royal Hospital.—H.S. for Fracture and Orthopædic Dept. At rate of £100.

Woolwich and District War Memorial Hospital, Shooter's-hill, S.E.—Res. M.O. £175.

The Chief Inspector of Factories announces a vacancy for a Certifying Factory Surgeon at Wigston, Leicestershire.

Births, Marriages, and Deaths

BIRTHS

BOLT.—On July 29th, at Great Russell-street, W.C., the wife of R. F. Bolt, M.R.C.S. Eng., of a son.

DAWES.—On August 11th, at Welbeck-street, W., the wife of W. A. Dawes, M.R.C.S. Eng., of a son.

FARNCOMBE.—On July 31st, at Euston-road, the wife of Ronald Farncombe, M.R.C.S. Eng., of a daughter.

GABRIEL.—On August 4th, at Highgate, the wife of W. B. Gabriel, F.R.C.S. Eng., of a son.

THOMAS.—On August 6th, the wife of Clement Price Thomas, F.R.C.S. Eng., of a son.

MARRIAGES

MACCALMAN—DAWSON.—On August 5th, at the Parish Church, Colonsay, Argyll, Douglas R. MacCalman, M.D. Glasg., to Helen Grego Dawson, M.A., elder daughter of Mr. John M. Dawson, M.A., LL.D., of Perth.

SWINBURNE—FENTON.—On August 6th, at Hawkhurst, Spearman Charles Swinburne, M.R.C.S. Eng., to Millicent, widow of G. R. Fenton, the Connaught Rangers, and elder daughter of the late Lieut.-Col. E. H. Montrésor, Royal Sussex Regt.

DEATHS

HAYNES.—On August 6th, at Leamington Spa, Frederic Harry Haynes, M.D., F.R.C.P. Lond., aged 90 years.

MACLEOD.—On August 8th, suddenly, at Edinburgh, William Bernera Macleod, M.B. Edin., Surgeon Captain, R.N. (retd.), aged 67 years.

PORTER.—On August 9th, at Wakefield, Surgeon Vice-Admiral Sir James Porter, K.C.B., K.C.M.G., M.B. Aberd., late Medical Director-General, Royal Navy, aged 83 years.

SPICER.—On August 8th, at Wimbledon Common, William Thomas Holmes Spicer, F.R.C.S. Eng., aged nearly 75.

WILSON.—On August 9th, at a nursing-home, Foden Wilson, M.R.C.S. Eng., of North Birkenhead, in his 72nd year.

WRIGHT.—At his residence, Boulderwood, Woking, after a brief illness, Alfred Wright, C.B.E., L.R.C.P. Edin., M.R.C.S. Eng., late Brevet Lt.-Colonel, R.A.M.C.

N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages, and Deaths.

NOTES, COMMENTS, AND ABSTRACTS

BENJAMIN BRODIE *

By H. E. MANSELL, B.M. Oxon., M.R.C.P. Lond.
LATE RESIDENT ASSISTANT PHYSICIAN, ST. GEORGE'S HOSPITAL

THE brightness of Hunter's star has tended rather to blind observers to other stars in the surgical firmament, but Benjamin Brodie's biographer and distant successor held Brodie to have brought the most acute intellect and the most powerful mind to the study of surgery, after John Hunter.

Brodie was born in 1783, the fourth of six children; his father, the Member of a Banffshire clan and a good classical scholar, held the living of Winterslow, near Salisbury. He sent none of his sons to school or to a university, but kept them well to their work at home. Of his home life Brodie writes in his autobiography:

> In the summer my brothers and myself rose at 6 o'clock and two hours were devoted to study (generally learning to repeat Greek and Latin poetry or Cicero's orations) before we breakfasted at 8 o'clock. Immediately after breakfast we resumed our studies. We dined at 3 o'clock and were then at our studies again from 4 to 6 o'clock. . . . On two days in the week, when my father was absent on public business, we had half-holidays. We had no other vacations during the whole year except on some grand occasions, such as a cricket match or the first few days of the skating season. . . . The result has been that the habit of being employed in some kind of study became a part and parcel of my nature. . . . Much of my success in my worldly career is, I am convinced, to be attributed to this discipline in my early years.

YOUNG DETERMINATION

The father impressed on his children that they would have to obtain their livelihood by their own exertions. For Benjamin the medical profession was chosen, and it is interesting to note that he disowned any particular liking for that profession and any faith in the special call to certain ways of life which some young men are supposed to have. He wrote that the persons who succeed best in professions are those who, having (perhaps from some accidental circumstance) been led to embark in them, persevere in their course as a matter of duty or because they have nothing better to do.

In a letter written in after-life to his elder son, then sixteen, he mentioned that his own four years between the ages of 16 and 20 were among the most valuable of his whole life.

> It was in that interval of time, he wrote, that I acquired habits of perseverance and industry, and that I learned to direct my attention to a particular object instead of travelling from one subject to another. I think too that I can recollect my having then, for the first time, meditated upon my own character and become sensible of some of my own faults, a branch of knowledge of more consequence than all the Greek and mathematics that schools and colleges can teach to the most zealous student. It is said to be desirable that you should know the world; but it is much more so that you should know yourself. Always keep before your mind the maxim which was inscribed over the Temple of Delphi: γνῶθι σεαυτόν.

After previously studying anatomy in the Hunter School in Great Windmill-street, he entered as a pupil of St. George's Hospital in 1803, and found that for himself at first, as for other students, all was confusion; the niceties of diagnosis, prognosis, and treatment were incomprehensible. The mist soon cleared, when every case became an interesting subject of inquiry. In his own vivid words "a great game was being played in which the stake was often neither more or less than the life or death of a fellow-creature, and in which those among the students who devoted themselves to their business performed a humble yet not unimportant part without any painful feeling of responsibility." Brodie gained some knowledge of materia medica, for by Dr. Baillie's advice, when he had no subject for dissection, he attended a chemist's shop at the corner of Little Newport-street. The proprietor of it, one Mr. Clifton, also practised as an apothecary, exercising his art among the tradesmen of the neighbourhood. His treatment of disease was simple. He had in his shop five large bottles—"Mistura Salina," "Mistura Cathartica," "Mistura Astringens," "Mistura Cinchonæ," and some kind of white emulsion for coughs, and out of these he prescribed for two-thirds of his patients. Brodie did not set this down to his discredit, for

> I have observed (he wrote) that while young members of the medical profession generally deal with a great variety of remedies, they generally discard the greater number of them as they grow older, till at last their treatment of disease becomes almost as simple as that of the Æsculapius of Little Newport-street. There are some indeed that form an exception to this general rule, who, even to the last, seem to think they have, or ought to have, a specific for everything, and are always making experiments with new remedies. The consequence is that they do not cure the patients, which the patients at last find out, and then they have no patients.

GOOD FRIENDS AND PROGRESS RAPID

In May, 1805, Brodie became house surgeon at the hospital, but, like Hunter, resigned the post after a few months in order to teach anatomy at the Windmill-street school. He also at this time accepted a proposal from Everard Home to act as assistant in his private practice and work for him at the museum of the Royal College of Surgeons of England. This latter employment proved to be of critical importance to him, not only because it kept his scientific outlook wide at a time when he might have been tempted to devote himself exclusively to practical surgery, but also because it led to an introduction to the great Sir Joseph Banks, who was president of the Royal Society from 1778 to 1820. At Banks's house he associated with Herschel, Davy, Woollaston, Young, Home, Abernethy, Carlisle, and others who formed the brilliant intellectual society of the day.

In 1808 Brodie was appointed "assistant surgeon to Everard Home"—not to the hospital, for the latter post was begotten later of the out-patient system, which, says Timothy Holmes, "lay like an incubus on all London hospitals." He became full surgeon in 1822, and held office till his resignation in 1840 for reasons which are well worth quoting:

> One of them was that I began to feel the necessity of diminishing the amount of my labours. Then I had long since formed a resolution that I would not have it said of myself, as I had heard it said of others, that I had retained a situation of such importance and responsibility when, either from age or indifference, I had ceased to be fully equal to the duties belonging to it; and lastly, when I saw intelligent and diligent and otherwise deserving young men waiting their turn to succeed to the hospital appointments, it seemed to me that there was something selfish in standing longer in their way, when as far as my own worldly interests were concerned I had obtained all that I could desire.

He was elected president of the Royal Medical and Chirurgical Society in 1839, of the Royal College of Surgeons of England in 1844, and of the Royal Society in 1858. He was the first president of the General Medical Council (1858–1860); president of the Ethnological Society in 1853, and president of the Section of Social Economy in the National Association for the Promotion of Social Service in 1857. He attended George IV., and in 1828 he was made surgeon-in-ordinary to the King. He was subsequently serjeant-surgeon to William IV. and

* Extracted from "Round the Board-room at St. George's," a paper read to the Osler Club on Feb. 22nd, 1935.

Queen Victoria, and in 1834 was made a baronet. His "Collected Works," which were edited by Charles Hawkins and published in 1865, contain his autobiography, numerous original contributions to surgical literature, including his masterly treatise "On Diseases of the Joints," and many introductory discourses and presidential addresses, which revealed the width and depth of his knowledge and interests. He died on Trafalgar Day, 1862, in his eightieth year.

PIONEER IN EDUCATION

Brodie revolutionised the teaching of surgery by his example at St. George's Hospital. Until his time the surgeons used only to go round the wards on two days in the week and never attended otherwise, except when there were operations to perform or when they were especially sent for on emergency. Brodie and Robert Keate, who was surgeon to the hospital from 1800 to 1853, were the first persons to be at their post daily, and there was never an urgent case that they did not visit in the evening and not infrequently early in the morning also. No wonder that the effect of so healthy a change was soon visible in the increase of zeal and diligence on the part of the students and in their increasing numbers. It was, in effect, the commencement of Brodie's reputation as a surgeon and teacher of surgery, and of the rise of the St. George's School to the high position which it held during the whole period of Brodie's services there. He also appointed clinical clerks and began to give clinical lectures, the first ever delivered in a London hospital. He always endeavoured to impress on the minds of his pupils the necessity of making and preserving written records of their experiences. To-day, when we hear so constantly of so much that is wrong with our medical curriculum, it is of interest to remember how bitterly Brodie complained that the great mass of students were so occupied by the large (and in his opinion unnecessary) number of lectures which they were required to attend, and in running from one classroom to another, that they really had neither the leisure nor the physical powers necessary for pursuing in any efficient manner the practical study of disease in the wards of the hospital. He considered it a great mistake to compare the examination of young men entering a profession with those for degrees in a university.

"The Senior Wrangler," he wrote, "might be a great mathematician and the first-class man in classics might be a first-rate Greek scholar, but the utmost that could be expected of a young lawyer or physician or surgeon was that he should show that he had laid such a foundation as might enable him to profit by the opportunities of experience presented to him afterwards. To be a thorough master of his profession in the beginning of his career was out of the question and was a thing to be attained only by unremitting study and close observation continued during a long series of years." These views were not applicable to the Fellowship of the College, which Brodie instituted "with the object of ensuring the introduction into the profession of a certain number of young men who might be qualified to maintain its scientific character, and would be fully equal to its higher duties as hospital surgeons, teachers, and improvers of physiological, pathological, and surgical science afterwards."

After Brodie's death a pupil wrote that Brodie was always distinguished for the value of the matter he had to communicate; those who heard him in the early part of his career found him then energetic rather than polished—struggling with the weight and mass of facts stored up in his mind—but in later years his delivery was fluent and perfect. And Sir Henry Acland, also a St. George's man, has said of Brodie, as a lecturer, that none who heard him could forget the graphic yet artless manner in which, sitting at his ease, he used to describe minutely what he had himself seen and done under circumstances of difficulty, and what under like circumstances he would do again or avoid. His instructions were illustrated by valuable pathological dissections which during many years he had amassed, and which he gave, during his lifetime, to his hospital. He considered it to be one of the most valuable and useful collections of the kind in the metropolis, and a similar view was expressed by Osler of the post-mortem records of the hospital, which have been continuous since 1841.

GREAT PUBLIC POSITION

Brodie held a unique position among his contemporaries, and his services were in wide demand. He was consulted by Brunel, the engineer of the Great Western Railway, when that gentleman had shown his inexperience as a conjuror by inhaling a half-sovereign. He attended Sir Robert Peel after his fall while riding up Constitution Hill, and has left a detailed account of the case. He was called as a medical witness in the celebrated trial of William Palmer, and later in the Smethurst case. Here his evidence was short, clear, and decisive, and a *Times* reporter added: "Sir Benjamin Brodie gave his evidence with great clearness—slowly, audibly, and distinctly—matters in which other medical men would do well to emulate such a distinguished example." Holmes, his biographer, comments:

> If all medical witnesses would follow Brodie's example, professional testimony would rise in public and in forensic estimation; we should cease to look on the degrading spectacle of witnesses who seem to be as much retained for one side or the other as the advocates are. They would speak of matters of fact, resting on the sure ground of their own and others' experience, avoiding dubious hypotheses, and the wranglings of pretended "science"; and, speaking in plain English, without bombast or technicality, they would guide and assist the court in forming a just judgment, instead of misleading and confusing it

Brodie's works repay perusal to-day for their variety of interest and for their qualities of clearness, common sense, and humour. A careful observer of life and all its functions, he studied death with equal care and minuteness, and it is consoling to read that "he had never known but two instances in which, in the act of dying, there were manifest indications of the fear of death." You will remember how Osler recorded from a study of about 500 death-beds, that the majority of the dying "gave no sign one way or the other; like their birth, their death was 'a hope and a forgetting.'"

Let me end this study of Brodie with another quotation from his autobiography:

> During these thirty-two years of office, the hospital, as far as my profession was concerned, was the greatest object of interest I possessed. Except during the brief intervals of my absence from London, it rarely happened that I was not at some time during the day within its walls. I was indebted to the opportunities which it afforded me for the best part which I had been able to attain. It had rendered my professional life one of agreeable study instead of one of mechanical and irksome drudgery. Some of my happiest hours were those during which I was occupied in the wards with my pupils around me, answering their inquiries, explaining the cases to them at the bedside of the patients, informing them as to the grounds on which I formed my diagnosis and my reasons for the treatment I employed, and not concealing from them my oversights and errors; and all this to kind and willing and only too partial listeners. My intercourse with the students, and I may add, with the patients also, was always to me a source of real gratification; and even now (many years afterwards) these scenes are often renewed to me at night, and events of which I have no recollection when awake come before me in my dreams.

THE RIGHT TO CURE

THE Institute of Identification at Rio de Janeiro publishes official archives containing contributions which are sometimes of more than merely forensic interest. For example, C. Brisard and M. Ducosté [1]

[1] Arquivos de Med. legal e Identificação, 1934, iv., 9.

both discuss the "right to cure"—more precisely the right of a surgeon to operate—without the consent or even against the will of the patient. The risk run by the surgeon who, in these circumstances decides to operate, of finding himself subsequently the victim of legal proceedings is minimised by neither author. This risk is naturally greatest if the patient dies, but even if recovery follows the patient or his friends may bring an action against the surgeon, especially if some residual disability remains. An instance of the latter would be a ventral hernia resulting from an abdominal section. Brisard quotes a case calculated to discourage well-meant interference. A man hanged himself; was found and cut down by a passer-by; in falling to the ground fractured his thigh; and successfully sued his rescuer for damages! In spite of this example, Brisard concludes that a surgeon must not shrink from exercising his own judgment should the occasion arise; consoling himself with the thought that unless negligence or faulty technique can be proved against him, he has good grounds for hoping that the court will take a lenient view of his case.

CANCER RESEARCH IN YORKSHIRE

THE annual report of the Yorkshire council of the British Empire Cancer Campaign relates work carried on at two research centres. Prof. R. D. Passey (Leeds) has continued his attempts to induce gastric cancer in rats by infecting them with the *Gongylonema neoplasticum* in the manner first described by Fibiger. Although the animals were successfully infected with a parasitic worm, and although its presence could be demonstrated in the œsophagus and stomach, in no case did cancer develop. This failure is remarkable as hitherto Fibiger's results have been accepted as generally valid.—Dr. Berenblum has continued his investigations on certain skin irritants, such as mustard gas, which have the power to inhibit experimental carcinogenesis by tar. He finds that, while all the compounds which inhibit carcinogenesis are skin irritants, not all skin irritants inhibit the carcinogenesis. With Drs. Orr and Kendal he has studied the action of mustard gas on cellular metabolism. It was found that anti-carcinogenic substances have the power of inhibiting glycolysis rather than respiration of tumour tissue. The substances related to mustard gas which do not inhibit carcinogenesis produce no effect on either respiration or glycolysis, or depress both equally.—Dr. K. J. Bonser, working with special strains of mice, has been able (following Lacassagne) to induce mammary cancer in male mice by repeated injection of œstrin. The positive results were obtained in male mice from a strain having a high incidence of spontaneous mammary cancer in the females. Differences in susceptibility to œstrin do not appear to be related to differences in the normal œstrous cycle.

Prof. E. Mellanby (Sheffield) has confirmed the observation that in a fowl bearing a Rous tumour the filtrable agent is distributed widely throughout the tissue and body fluids. Experiments were carried out in which the experimental induction of cancer in mice by dibenzanthracene was compared with that in mice similarly treated but receiving at the same time injections of the Rous agent from fowls. The results show that the introduction into mice of the Rous agent from the fowl did not significantly modify the development of cancer in mice. Experiments bearing on the chemotherapy of cancer were carried out with the idea of finding substances which would interfere with the respiration of cancer cells. Two such substances were investigated, namely, dilute potassium permanganate and various snake venoms.—Prof. H. W. Florey also submits a short report on various new lines of investigation.

AN INSULIN POCKET-CASE

THE "Wellcome" brand insulin pocket-case (No. 46), made of nickel-plated metal, is specially designed by its measurements to fit comfortably into the pocket. It contains the materials and apparatus necessary for the administration of insulin. The contents are as follows: (1) "Agla" insulin syringe (1 c.cm. graduated in 1/20ths). (2) "Agla" surgical needle (rustless steel), in glass-stoppered container. The container is intended for ethyl alcohol, in which the needle will be immersed, thus maintaining it in an aseptic condition. (3) 5 c.cm. rubber-capped

phial of "Wellcome" brand insulin, containing 100 units. (4) "Vaporole" iodine applicator for use when sterilising the skin at the site of injection and the rubber cap on the insulin phial prior to removal of a dose.

The outfit should be specially useful when injections are to be self-administered.

NEW PREPARATIONS

THEOTONE.—This preparation is manufactured in tablet form by Messrs. Allen and Hanburys Ltd., London, the formula being theobromine grs. 5, phenobarbitone gr. ¼. The diuretic action of theobromine has been investigated and established for some years. While it is also a dilator of the coronary arteries, it resembles caffeine in its effects, but its use is apparently not contra-indicated when the blood pressure is high. Its trial in cases of angina pectoris or intermittent claudication before trying sympathectomy has been advocated by certain medical observers. The combination of theobromine with phenobarbitone has been found beneficial in cases of high blood pressure and conditions which may be summarised as those of cardiovascular overactivity.

SALEPSI RUSSI.—This preparation, which can be obtained in solution and in pill form, is designed as a sedative, and claims to have special indications in epilepsy. It is manufactured at the chemical and pharmaceutical works of Russi and Co., Antona, Italy, Messrs. Coates and Cooper Ltd., London, being the English agents. The formula is: 25 drops (1 c.cm.) contain ethyl-phenyl-barbituric acid, 0·05 g.; cascara sag., 0·10 g.; antispasmodic extracts, scopola, stramonium; valerian, 0·04 g.; saccharin, 0·002 g. Messrs. Coates and Cooper inform our readers that specimens will be forwarded upon request.

THE DEATH OF JINNY.—To our issue of July 27th Dr. Kathleen Vaughan communicated a note upon a pet chimpanzee named Jinny, for whom a home was needed. Dr. Vaughan's note brought numerous communications to Jinny's owner, Mrs. Basham, but sad to relate Jinny died on the Monday following the publication of her story. We learn that many offers to receive her as an inmate were received from readers of THE LANCET, and the news of her death is announced here as an explanation why Mrs. Basham has not replied to all the letters.